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THE TEACHING OF OPHTHALMOLOGY IN THIS COUNTRY

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DETROIT

An attempt was made by the International Congress of Ophthalmology in 1929 to gather information concerning the method of presenting the subject of ophthalmology to undergraduate and postgraduate students in all the countries of the world, and I was assigned the task of collecting the data from England and her dominions and from North and South America. With your permission I shall draw on some of the material collected for that report. In this country, letters were sent to the colleges and universities in "Class A" group as graded by the American Medical Association, requesting information on the number of hours devoted to the teaching of ophthalmology, together with the method of presenting the subject before the undergraduate students. Replies were received from forty colleges and universities. From these data the following facts were assembled: In six institutions ophthalmology is not taught as a special subject. In the remaining thirty-four, the average number of lecture hours is twenty; of clinical and demonstration hours, forty-four, the average total being sixty-four hours. In two institutions (Yale University and the College of Long Island) no lectures are given on ophthalmology, all the instruction being in demonstration courses. Yale gives thirty-six hours and the College of Long Island twenty-four. The most striking revelation was the fact that the lecture hours varied from none to forty-eight, and the clinical and demonstration hours from eight to one hundred and twenty.

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Most institutions give the clinical instruction and demonstrations in the last year of the medical course; a few in the third, or third and fourth years. The number of subjects included in the course of instruction in the United States shows but slight variation. In two institutions ocular pathology is given as a part of the demonstration course, but for the most part only the use of the ophthalmoscope and the bearing of ophthalmology on general medicine, together with the recognition and treatment of the more common diseases of the eye and its appendages, are included in the courses given. In only a few institutions is the teaching of accurate refraction attempted, although the importance of a careful refraction for the relief of symptoms is emphasized in nearly all reports. The lecture-demonstration is generally advocated as the best method of presenting the subject to the undergraduate student. Assignment of cases to be worked up and presented by the student before the section is also thought to be a most effective method of teaching. Information concerning the frequency of quizzes and examinations was not requested in the questionnaire, but voluntary information advocating both the quiz and the examination came from the majority of the institutions.

The necessity for teaching the essentials of the various special branches of medicine is generally accepted, but there is still a difference of opinion as to the best method of presenting the subjects and of administering the departments. There are no laws or statutes in the United States requiring a degree in ophthalmology for the treatment of diseases of the eye. Any licensed practitioner of medicine may prescribe for his ophthalmic patients if he so desires. The Council of Medical Education of the American Medical Association recommends that the minimum number of hours devoted to ophthalmology be fifty, divided as follows:

Lectures	15
Clinical	15
Section work in hospital and dispensary	20

The Medical Examination Boards in the several states may establish certain requirements for registration, and by naming the minimum hours required influence the teaching of medicine in the individual states. In eighteen states ophthalmology is not specifically required for medical registration; in seven it is required, but the number of hours of preparation is not stated, while in twenty-three the recommendation of the Council of Medical Education of the American Medical Association is specifically stated.

The United States has no monopoly on the variation in the number of hours assigned to ophthalmology, as shown by the accompanying table.

From the table it can be determined that the average number of lecture hours in the six countries in which lectures in ophthalmology are a part of the regular medical program is sixteen, and the average number of demonstration hours is seventy-one. The average total number of hours in the one hundred and eight institutions in the eleven countries included in this report is sixty-nine.

All special departments in a medical curriculum have a twofold function.

1. To establish the relation between the work given by the special department and that required in the general training.
2. To familiarize the student with the methods of diagnosis and treatment of the more common diseases seen clinically in each department.

There is a great difference between the training of specialists and the teaching of the essentials of the special branches of medicine and

Country	Number of Institutions	Lecture Hours		Clinical Hours		Average		Average Total
		Maximum	Minimum	Maximum	Minimum	Lecture	Clinic	
Great Britain	12	24	10	150	30	14.0	77	91.0
Australia	2	10	9	80	18	9.5	49	58.5
Canada	7	60	10	60	13	22.0	34	56.0
Egypt	1	24	..	38	..	....	..	62.0
India	19	65	10	283	24	24.0	155	139.0
New Zealand	1	40	..	30	..	....	..	70.0
Siam	1	12	..	12	..	....	..	24.0
South Africa	1	16	..	20	..	....	..	36.0
South America	3	12	12	144	24	12.0	68	70.0
United States	40	48	4	120	8	19.0	46	65.0
Philippine Islands	1	34	..	...	58	....	..	92.0

surgery to the undergraduate, and I agree entirely with those who contend that the undergraduate teaching of the specialties should be limited to the requirements necessary for a thorough general medical training, and that the development of specialists should be left for postgraduate work. A reasonable time, however, must be provided for instruction in the special branches of medicine and surgery, if students are to be given a thorough training for the general practice of medicine.

Some of the technical subjects can be taught just as well, perhaps better, by assistants, but the broad aspect of each specialty in its relation to a general medical training can be adequately presented only by one of wide clinical experience in that specialty. Besides contributing to the undergraduate program, a properly organized special department should be so administered as to encourage the spirit of research in the members of its staff and to serve, through scientific contributions, the institution of which it is an integral part.

While there is a great discrepancy in the number and disposition of hours allotted to ophthalmology in the various institutions, it would seem that some assignment could be made that would result in a more

balanced curriculum and at the same time offer a practical course for the proper presentation of the subject. In my portion of the report made before the International Congress of Ophthalmology it was recommended that a minimum of sixty hours be devoted to ophthalmology, fifteen lecture-demonstration hours and forty-five clinical teaching hours. One great difficulty, however, is that balancing the curriculum does not equalize the ability of the various teachers to present the subject. At different periods of time certain institutions have stood out preeminently, and one hears of the wonderful faculties of the eighties and the nineties. The curriculum was the same both before and after these periods of distinction. Why did these institutions stand out so prominently at such times? It was undoubtedly due to the efficiency of the teachers on the various faculties.

The correlation of the work offered by each department is quite as important as the standardization of the number of hours devoted to each specialty. There are, in general, two possible systems for interdepartmental correlation, namely, the refer system and the conference system. While the latter is more ideal, the element of time makes it inconvenient or impossible, and I believe that for the routine work of a general hospital the refer system offers the much more practical plan and that only the occasional case should be examined in conference. The objection to the conference system, as applied clinically, does not hold to the same extent in the teaching plan, however given, and it is possible, as suggested by de Schweinitz, that special lectures and demonstrations could be interspersed in the courses in medicine when points of intimate contact occur. For example, when students are engaged with cardiovascular and renal diseases, the practical application of ophthalmic examinations might be given. The same coordinated work with surgery when intracranial pressure is under discussion is equally possible.

The increasing appreciation of the dependence of ophthalmology on the general medical examination is well shown by the passing of the isolated eye hospital. To meet this situation the special hospitals are either adding a staff representing the essential branches of medicine or are becoming an integral part of the medical center.

In many institutions the recognition of the importance of certain specialties has resulted in the establishment of separate departments. Just what determines when a branch of medicine or surgery should be given a special department is difficult to say. Apparently, the departments that have been so recognized in recent times have been brought about through the preeminence of individual workers and not through any sympathetic plan on the part of the governing bodies. I think, however, that all will agree that more progress has been made in every instance than would have been the case if the departments had remained submerged.

POSTGRADUATE INSTRUCTION

The question of postgraduate instruction in this country is somewhat difficult to evaluate. Doubtless many internships in hospitals and assistantships on clinics offer efficient training in ophthalmology, but for the purposes of this communication only those centers that offer systematic instruction in addition to the clinical training will be considered.

There are, in the United States, seven institutions that give postgraduate instruction in ophthalmology, namely, the University of Colorado, the University of Iowa, Johns Hopkins University, The Massachusetts Eye and Ear Infirmary, the University of Michigan, the University of Minnesota and the University of Pennsylvania. A brief outline of the work offered in each instance is as follows: A course in ophthalmology leading to a graduate degree is given at the University of Colorado. The departments of physics and anatomy, also the eye clinic and teachers in the undergraduate medical department are utilized to give instruction. A twelve weeks' intensive course is given. The remainder of the year is devoted to supervised study of ophthalmology and the preparation of a thesis. Others are admitted to the course, but a candidate for the degree is required to be a registered physician, to have had a year's internship in a general hospital, or its equivalent in laboratory work, and to possess a preliminary knowledge of mathematics. A degree of Master of Science in Ophthalmology is conferred at the discretion of the faculty. A course limited to eight graduates is offered at Iowa, each student serving two years as a member of the hospital staff. The work is mostly clinical, although three hours a week are devoted to lectures in other departments. Johns Hopkins University offers special instruction in ophthalmology to eight graduate residents and interns (four of each). No fixed time is required for service of the residents. A limited number of students (never more than four) may be taken for postgraduate work. The Massachusetts Eye and Ear Infirmary offers a postgraduate course in ophthalmology by which six interns are given definite instruction during the eighteen months' service at the infirmary as house officers. The course covers nine months. Each intern is required to devote three hours (one evening a week) to an exercise that is arranged for him. The postgraduate teaching of ophthalmology at the University of Michigan is limited to three graduates, each serving as a member of the staff for three years, after one year in a general hospital. The service is mostly practical, although systematic lectures or demonstrations are given in embryology, anatomy, pathology and physiologic optics. The senior member of the staff conducts the quiz given to the students in the sections of the junior medical class, and all the staff assist in the demonstration of cases before the members of the section of the senior class.

Those who hold a collegiate degree are eligible to register in the postgraduate school for a master's degree. Otherwise, no certificates or degrees are granted. Fellowships in ophthalmology are granted at the University of Minnesota and the associated Mayo Clinic. The holders of these fellowships receive in the first year systematic instruction in fundamental branches and clinical work. In the second and third years the time is devoted to clinical work, teaching and research in the preparation of a thesis. Completion of this course entitles the Fellow to the degree of Master or Doctor of Science in Ophthalmology. At the University of Pennsylvania a two year course in ophthalmology is offered, limited to sixteen students. The course consists of systematic instruction for eight months by means of didactic and clinical work. All the subjects and correlated subjects are included in the course, which is so arranged that the various branches are introduced in a sequence corresponding to the progress of the student. The course is given by a staff of forty-six physicians, thirty-two of whom are specialists in ophthalmology. About six hours' formal instruction per day is provided for five days of the week, with laboratory work and clinics on Saturdays. A certificate is granted at the successful completion of the first year, and indicates satisfactory study of the principles underlying practice in ophthalmology and eligibility to the second year clinical work. No systematic didactic lectures are given in the second year course, which includes one thousand and fifteen hours directed specifically to the authorized subjects and the preparation of a thesis. The work is supervised by a preceptor and may be executed either intramurally or extramurally. The student physician is required to make a detailed weekly report of the progress of his work. The report includes the number of hours devoted to each subject by himself and by his teacher, also the number of cases seen and a list of the diseases demonstrated. This report is endorsed by the preceptor and forwarded to the dean, who, in this manner, has a check on both the student and the teacher. The degree of Master of Medical Science is conferred after the successful completion of a second year in a clinical department. A third year term of definitely assigned research work may be arranged, on the successful completion of which the degree of Doctor of Medical Science may be conferred.

There are several postgraduate courses given by schools and clinics that are not included in this report, the length of the course varying from ten weeks to five months. The clinical work offered may be of a high order, but the weak point in most of them is that no entrance restrictions are enforced, and the fundamental subjects are seldom adequately taught.

In presenting an outline of the "Teaching of Ophthalmology in This Country," I should be remiss if I did not refer to the activities

of the American Board for Ophthalmic Examinations. The object of the Board is to examine candidates and to certify as to their fitness to practice ophthalmology, and to act in an advisory capacity when requested. There are nine members on the Board, three from each of the national societies, The American Ophthalmological Society, the Section on Ophthalmology of the American Medical Association and the Academy of Ophthalmology and Oto-Laryngology. Each society elects one member annually to serve for a term or three years. No member is permitted to serve more than two consecutive terms, but may be reelected any time after the expiration of one year off the Board.

The Board was organized in 1916, and has held regular meetings since that time. In all, nine hundred and ninety-nine candidates have been certificated. The importance of the certificate to the holder has been greatly enhanced by the fact that not only the national societies have made the certificate part of their requirements for membership, but several hospitals require it for positions on the staff. The efforts of the Board have only an indirect effect on the teaching of ophthalmology. The members of the committee recommend that two years be the minimum for training. Of the two years, the first should be devoted to systematic work divided between fundamental and clinical subjects; the second should be spent as an intern in a hospital, as assistant in a well organized eye clinic, or as a private assistant in the service of a competent ophthalmologist. A list of the subjects to be included in the fundamental and clinical courses is recommended by the Board, but no course of instruction is attempted. In addition to the two years recommended for training, a third year in the practice of ophthalmology, or as an assistant, is required before the candidate shall be eligible for examination by the Board. Each candidate is required to submit case records and to show an intimate knowledge of the following subjects:

1. External diseases of the eye
2. Ophthalmoscopy
3. Pathology and anatomy
4. Errors of refraction
5. Ocular muscles
6. Perimetry
7. Relation of eye conditions to general disease
8. Therapeutics and operations

I have served for several terms on the Board and have never had a more satisfactory committee assignment. Usually the whole membership is present at the meetings, and the duties of the Board are performed in the most painstaking and conscientious manner. Dr. William H. Wilder, of Chicago, has been the secretary and treasurer since the time of its organization, and to his untiring effort and good judgment

is due much of the success of the work. That the work of the Board has been appreciated is attested by the fact that similar organizations have been instituted for otolaryngology, obstetrics and gynecology, dermatology and radiology, and there are now under way plans for similar boards for proctology, neurology and psychology and orthopedics.

If I were asked to mention the most interesting situation that has presented itself during my teaching experience of over twenty-five years, it would be the great gulf that exists between the radical educator on the one hand, and the ultraconservative teacher on the other. Fortunately, the more radical the views at either extreme, the fewer the apostles, and as one approaches the conservative mean, the number of advocates increases. Therein lies our safety, the right and left parties offering the suggestions and objections, the middle party doing the conservative, constructive work. Perhaps this accounts for the stability of the medical curriculum. Attacked on all sides, it has maintained a sturdy resistance that excites admiration and gives promise that it may survive the revolution.

VASCULARIZATION OF THE ANTERIOR SEGMENT OF THE EYE

THE BEARING OF THESE STUDIES ON SOME OPERATIVE PROCEDURES, INCLUDING A POSSIBLE SUPPLEMENTARY PROCEDURE FOR GLAUCOMA; PRELIMINARY REPORT

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This study developed from observations on a case of exaggerated pinguecula with great vascularization. Such was the aggravated condition of the multiple pingueculae, with the accompanying vascularization, that the entire visible conjunctiva of the palpebral opening on every side was highly congested and discolored so that the patient, a woman of 38 years, hesitated to appear in public. This condition was of many years' duration. When local remedies and astringents failed to eliminate the trouble, I turned to an operative procedure.

Superficial inspection designated the source of the vascular supply to the pinguecular areas as coming from the conjunctival vessels, which were much enlarged and could be seen after their branching to terminate in separate pinguecular areas. Therefore I felt that if these vessels could be obliterated at their base, the redness and vascularization, which were the most unsightly feature of the case, and also the pingueculae would be, in a measure, dissipated.

Consequently, the procedure at first adopted was the liberal removal of a strip of conjunctiva at the canthi. This was extended vertically for about 8 mm. in length and 3 mm. in breadth, and seemingly obliterated all the conjunctival supply.

Higher magnification showed, however, that a separate vascular supply to numerous pingueculae still existed. It was therefore evident that many of the pingueculae were supplied from a source other than the conjunctival vessels, from in fact the entirely separate and deeper reticulum of ciliary vessels, and it was evident that a peridectomy or section of the conjunctiva at the canthi did not and could only partially affect them, and that cutting off the conjunctival vessels as a means of obliterating the vascularization contributed by this secondary system was ineffectual.

The procedure further adopted, therefore, was to make a pocket dissection, separating the conjunctiva from the original resection at the canthi as far as the sclerocorneal margin, and then to curet

the surface of the muscle and the under surface of the conjunctiva thoroughly throughout the entire affected area to the corneal margin. This was followed by profuse hemorrhage, which was easily controlled. The blood clots, however, raised the conjunctiva 2 or 3 mm., so that when the operation was finished, the picture was one of extreme pocket subconjunctival hemorrhage or hematoma. The conjunctival openings at the canthi were not sutured.

The patient made a perfect recovery without any untoward incident. The vascularization and redness were 95 per cent dissipated, though very small areas of pingueculae remained; now, six months after the operation, the condition has resulted in almost a complete cure, most satisfactory to both the patient and the operator.

The obvious deduction from this case was that the vessel supplies came from two sources, and that to trust the severing of the conjunctival vascular system alone to obliterate vascularization of the anterior segment of the eye would be futile unless both the conjunctival and the deeper ciliary supplies were dissipated.

Profiting by this first experience, a second study was instituted to determine whether in the instance of a vascular fasciculus the vessels were altogether composed of the conjunctival system alone or of both systems. Overwhelming evidence was observed that quite as large a portion of the vascular contribution came from the ciliary system (the deeper) as from the conjunctival, as the vessels from this deeper system passed directly under the membrane (capsule) into the corneal substance.

Also on inspection before operation the two systems of vessels, the ciliary and conjunctival, did not seemingly anastomose except at their terminals, were distinctly separate and, by obliteration pressure tests, were both proved, at least in large measure, to be arterial. The ciliary reticulum of vessels did not, as in the case of pinguecula, come to the surface and could be distinctly traced under Tenon's capsule to the corneal margin, where they independently penetrated the cornea and contributed to its vascularization.

This second case in which I operated to determine the sources of vascularization of the cornea was one referred by Dr. John Evans. In this case a pannus had been present over a period of sixteen years and a tarsal resection had been performed; while numerous areas of vascularization existed in the cornea, one particular fasciculus had been ligated two or three times without cure.

In this case I performed a complete peridectomy, removing a section of conjunctiva around the corneal margin, about 2.5 mm. in diameter, and completely severing and cutting off all the conjunctival vessel supply. The even removal of this section was accomplished accurately and satisfactorily by circling the cornea with a narrow punch, which

clipped a furrow as the conjunctival edge was elevated with a tooth forceps. Incidentally, if the operator, after doing this, finds that the conjunctiva pulls away toward the fornix, greatly broadening the exposure and section, he may bring the conjunctival edge back to the corneal margin by introducing a purse-string suture, thus closing the gap to the desired breadth.

To resume, the conjunctiva in this case, being retracted backward to an area 6 or 8 mm. from the corneal margin, revealed the reticulum of ciliary vessels absolutely intact and not influenced by the conjunctival peridectomy. These could be seen passing directly into the superficial cornea or the remnant of Bowman's membrane. In an effort to obliterate them, I used curettage and grattage, both of which were disappointingly unsuccessful.

Higher magnification again showed that the reticulum of deeper ciliary vessels had not been damaged to any great extent by the curettage. There was no bleeding from them, and they presented the appearance of the mesentery of a frog, the vessels of which had been injured by irritation but not destroyed. In other words, the deeper vessels had not been obliterated but only traumatized.

After the curettage failed, I felt that further surgical intervention on this eye was not advisable for the time being. The destruction of the ciliary system was therefore postponed.

This incident of failure of curettage and grattage, however, disclosed a strikingly practical and valuable point. The reason for their failure to destroy fully the second system, as seen by exposure and resistance to the curet, was that these deeper ciliary vessels were covered by a glossy resistive translucent membrane. This was probably Tenon's capsule which they passed under. This is so intimately adherent to the sclera in an area of 5 mm. from the limbus that it cannot be separated. Fuchs[1] gives a vivid description of these systems and Leber a clear diagrammatic illustration of the deeper ciliary reticulum.

I would supplement their excellent anatomic outline by an observation gained directly from surgical experience, *that the opaque dim violet hue of this ciliary reticulum is not so much attributable to its depth as to the dense translucent membrane under which it passes and is confined.* This membrane completely and distinctly separated them from the more superficial conjunctival system and, as I said, accounted for their pale, less distinct, semiopaque and translucent appearance before reaching the cornea, in contrast to the more vivid conjunctival vessels, which are much brighter in hue. These conditions, together with the fact that the conjunctival vessels are movable (as can be easily ascertained by passing the

1. Fuchs: Textbook of Ophthalmology, translated and edited by Alexander Duane, ed. 6, Philadelphia, J. B. Lippincott Company, 1919, p. 138.

finger over the lid, making pressure and ruffling the conjunctiva) and the deeper vessels stationary, plainly make possible the differentiation.

Thus the defeated efforts at curettage in this second case pointed to the conclusion that the covering membrane must be dissipated in order that the secondary and deeper vessel supply be destroyed. It was demonstrated that this could best be accomplished by multiple linear incisions in the third, the following case.

The third study of this question as to the surgical treatment of vascularization of the anterior segment was made on a case of secondary glaucoma in which enucleation was advised and undertaken.

Before enucleation I separated the conjunctiva from the corneal margin in the usual manner and freely retracted it back behind the equator. The conjunctival system was thus eliminated, and a highly engorged secondary system of ciliary vessels was perceived, slightly elevated and seemingly under pressure. Again in an attempt to destroy the ciliary vessels, grattage was used wtih the same result, and only moderate damage was done to this deeper system. The vessels did not bleed, but became dilated and engorged. As I realized that to obliterate this ciliary reticulum the covering membrane would have to be destroyed, a von Graefe cataract knife was used, and in an area of about 10 mm. square about seven parallel linear incisions were carried down to the sclera, incising the membrane and sectioning every vessel within the 10 mm. area.

The immediate picture of obliteration of the vessels was spectacular. The vessels bled freely, and after the débris was sponged away the sclera was white and glistening, with no perceptible vessels. No vascularization remained within the incised area. This section of destruction was in striking contrast to the adjoining reticulum, which remained highly congested and vascularized and was quite intact and covered by its protective membrane.

The striking picture in this third case was the greatly engorged reticulum of vessels evidently confined and under pressure of the dense membrane already alluded to, the profuse hemorrhage from them when the membrane was incised and they were sectioned by linear incisions, and the resistance of both membrane and vessels until these incisions were made.

With this preliminary report I would also append the fact that further surgical studies bearing on some of the chronic inflammations with vascularized corneas and on the question of tension are in progress. It is within the range of possibility that by the complete obliteration of this underlying vessel system over a considerable circumferential area, which can be easily reached, reduction of tension may be achieved by depletion of the blood supply of the interior and anterior segment of the eye.

If so, the procedure thus far outlined would be a complete peridectomy, the turning back of the conjunctiva to the equator, multiple linear incisions, 2 mm. apart, for an area extending 5 or 6 mm. or even farther posterior to the corneal margin, and the replacement of the conjunctiva, when this is accomplished, to a position at or near the corneal margin by the introduction of a purse-string suture.

The practical value of the observations made in these preliminary studies is as follows:

1. A peritomy, a peridectomy or ligation of the conjunctiva undertaken for the cure of corneal vascularization which does not take into account the obliteration of the deeper ciliary reticulum may absolutely fail by reason of the fact that the vessel supply of this deeper ciliary system has not been touched.

2. If it is desired to destroy the secondary reticulum, curettage or grattage is not likely to be wholly successful, as these deeper ciliary vessels are covered by a glistening, superimposed, strongly protective, resisting membrane, probably Tenon's capsule, which distinctly separates them from the conjunctival system. The best way to destroy them is by linear multiple section of both membrane and vessels over a considerable prescribed area.

3. These studies show the value of associating descriptive anatomic relationship with practical surgical experiences, as from such observations in my work on this subject were developed the following operative procedures: (1) an operation for subconjunctival excrescences which leaves the conjunctiva intact; (2) an exact sectional method for a peridectomy; (3) destruction of the secondary system of ciliary vessels; (4) the application of this last procedure for the depletion of a large portion of the ciliary blood supply.

After six months investigation, I made a report on this subject at the October, 1932, meeting of the New York Ophthalmological Society, and I hope in the near future to present drawings, microscopic sections, slit-lamp illustrations and further progress in this study.

ADDENDA

Since the first portion of this preliminary report was written, my attention has been concentrated on the influence that depletion of the swollen and inflamed anterior ciliary reticulum would have on the question of pain, tension and chronic ciliary congestion. This was stimulated by the picture of immediate depletion of an engorged ciliary area obtained by multiple linear incisions, already portrayed in the third case which I described.

Some difficulty has attended this study: first, on account of the natural reluctance to use sufferers from glaucoma as material for experi-

mental work, and second, because it is difficult to determine how much benefit is due to an anterior vessel ciliarotomy when it is done in conjunction with iridectomy, trephine and the Lagrange operation.

In the following cases, however, anterior vessel ciliarotomy alone was performed on two patients, and in another patient, with acute glaucoma, a partial ciliarotomy was performed in conjunction with a conjunctival flap iridectomy and a modified Lagrange operation.

CASE 1.—A. T., aged 58, an Italian, had a condition diagnosed as acute glaucoma. He was averse to operation, but later consented to the lesser procedure. An indefinite history of onset was given; the patient had had a painful eye for five weeks preceding his admission to the hospital. He received no definite medical treatment during this period. Vision in the right eye was 20/20; in the left, it amounted to bare perception of light. Tension on admission was normal in the right eye and 90 (Schiötz) in the left; it was reduced to 50 in the left eye after using pilocarpine 1 per cent, physiostiamine 0.5 per cent and dionin 5 per cent for five days, and then remained stationary. The cornea was slightly hazy. An engorged reticulum was marked. The pupil was moderately dilated. Pain continued unabated.

Operation.—After turning back the conjunctiva, complete vessel ciliarotomy was performed on the right eye. The reticulum was thoroughly sectioned by linear incisions, 1.5 mm. apart, by a von Graefe knife carried through the membrane in an area of about 6 mm. surrounding the limbus. The engorged reticulum completely subsided after evacuation. When the blood was sponged away the sclera was white and clear to the limbus within the area sectioned. Punctate hemorrhages were noticeable on the surface. Bleeding was of short duration. The conjunctiva was closed with a purse-string suture.

Observations.—The tension before the operation was 50; immediately after operation, 35. Ten days later, at the present writing, it is still 35. The pain, which had been excessive and continuous for five weeks, immediately subsided, and this condition has been maintained. The pupil was moderately contracted. The reaction was moderate, and the patient is making a good recovery. Vision is slightly improved. This patient will probably undergo an iridectomy if he will consent.

While it is fair to state that after operation the patient used a solution of pilocarpine (8 grains [0.5 Gm.] to the ounce [28.3 Gm.]), it is nevertheless noteworthy that before operation the tension was 50 and at the time of operation the drop of 15 occurred, that the ciliary reticulum engorgement was completely dissipated and also that pain, which had been excessive for five weeks, ceased from the time of operation. Vision was slightly improved and the pupil smaller.

CASE 2.—E. A., an Italian woman, aged 63, gave a history of diabetes with vitreous hemorrhages. Before admission to the hospital, on Nov. 28, 1932, she had initial attacks of glaucoma—in the right eye in June, 1932, with a tension of 90; in the left eye in September, 1932, with a tension of 80—at which times iridectomy had been performed, but the tension returned.

On November 28, when the case was referred to me by Dr. Rudolph Cutino, the tension was 90 in the right eye and 40 in the left. Both eyes had been continuously painful for two months. Both were blind and enucleation had been advised but not accepted.

Operation.—On the right eye no ciliarotomy was performed but posterior trephines were done, two of which tapped the region above the choroid, one com-

pletely draining the vitreous. Tension was subnormal immediately after operation, and at the present writing, ten days after operation, is 18.

In the left eye, on which my greater interest was concentrated, and on which iridectomy had been previously performed in June, 1932, small petechial hemorrhages of the iris existed. The pupil was moderately contracted, the cornea clear and pain severe. A markedly engorged ciliary reticulum existed.

Ciliarotomy was performed as previously described. Tension before operation was 40; immediately after operation it registered 20. Ten days later it was 18. The pain had completely subsided. The reaction was moderate.

Observations.—Anterior ciliarotomy alone was performed on the left eye. The tension dropped to 20 at the time of operation. Pain, which had been excessive for two months, disappeared from the time of operation. Tension has been maintained at 18.

Case 3.—A. B., an American, aged 52, had no history of glaucoma. However, one day preceeding the attack, he experienced a dull ache over the temple and malar region on the right side. On the night of December 6, he woke with violent pain in the right eye. Early on the morning of December 7, he was referred to me. In the right eye vision amounted to perception of fingers at 3 feet; in the left eye it was 20/20 + 4.25 sph. The right cornea was steamy, the fundus invisible and the pupil dilated. Tension in the right eye was 80; in the left eye, normal. The patient was nauseated and in semicollapse.

Operation.—The patient was admitted to the hospital the same day. An operation of combined iridectomy and the Lagrange procedure, with additional ciliarotomy, confined to the exposed flap area, was performed. The ciliary reticulum was notably swollen and engorged. Tension fell to normal and was subnormal eleven days after operation.

Observations.—The same picture of ciliary vessel depletion was observed as in the other cases. The use of the combined procedure, followed by no undue reaction, points to the feasibility of combining ciliary depletion by ciliarotomy with other operations in the area of conjunctival exposure. In this case it is impossible to estimate the contribution in reduction of tension by the ciliarotomy other than by the noticeable dissipation of the reticulum at the time of operation.

CONCLUSIONS

Interest centered on the question of the best manner of doing the ciliarotomy, and I believe it desirable to expose the ciliary reticulum by dissecting back the conjunctiva. From experience I know that curettage, grattage and scarification of the reticulum are not satisfactory. Dissection and separation of the confining membrane (capsule) in this region are almost impossible, so intimately is it blended with the sclera. The actual cautery will defeat the object to be obtained by sealing the vessels and perivascular spaces. Multiple section of both membrane and ciliary reticulum, as described, with a sharp von Graefe knife I have found very satisfactory. It should be realized that the cases in which the operation was performed were in the category of extreme glaucoma, wherein the anatomy was pathologically, and in two instances chronically, blocked.

Among the benefits resulting from the ciliarotomy, it is reasonable to suppose that one may obtain not alone depletion of the ciliary vessels but some lymph drainage from the multiple perivascular lymph spaces, rendered patulous by the procedure. It is striking that though both the conjunctival and the anterior ciliary blood supply had been removed, the clarity and luster of the cornea were not impaired but improved. Also, after the ciliary reticulum was dissipated, the area did not present any appearance of thickening, inflammation (scleritis) or superimposed inflammatory tissue. This indicates that the usual circumcorneal ring of injection is in reality engorged reticulum rather than ciliary inflammation imparted by continuity from the ciliary body through the sclera.

The fact that the procedure relieved the pain was indicative that the confined pressure of this vessel reticulum, when dissipated, depleted the ciliary body. The ease with which at least half of the ciliary area can be depleted in the conjunctival flap trephine and iridectomy operations makes the procedure available as a supplementary procedure. *It is just possible that the addition of this procedure of depletion of blood and extirpation of vessels in the ciliary area and the moderate supplementary drainage that it offers may be the means of preventing postoperative tension, so often noticeable after well regulated operations for glaucoma.*

I realize that the question of reduction of tension must receive further confirmation from a larger and more varied number of cases. However, in balancing the foregoing experiences, I cannot but feel that anterior vessel ciliarotomy, properly performed, may in the future occupy at least a definite supplementary place in the relief of congestive glaucoma, also in the relief of pain in chronic cyclitis and possibly in simple glaucoma when vessel stasis is exhibited by exaggerated vascularization.

This conviction is based not so much on the reduction of tension obtained in the two cases in which the procedure was exclusively done as on the exhibition of the depletion of the ciliary reticulum which was amply and, I may say, spectacularly demonstrated, and which must of necessity relieve and drain the ciliary body. The witnessing of this result is convincing.

LIPEMIA RETINALIS DUE TO DIABETES MELLITUS

WITH REPORT OF A CASE

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After a careful review of the literature to date, we found that there were only forty cases of lipemia retinalis reported. The first case was reported by Heyl in 1880. Gray and Root described two cases in 1923, from Joslin's diabetic clinic at the New England Deaconess Hospital. Since then, to our knowledge, only two other cases were observed at this institution (McKee and Rabinowitsch). In spite of routine ophthalmoscopic examination of the fundus oculi in all diabetic patients at the Morrisania City Hospital, this is the first case in our service of lipemia retinalis in diabetes.

The relation of lipemia retinalis to lipemia, and of both to fat metabolism, is as yet an unsettled problem. The available literature on the subject does not yield any clear facts in regard to the varying phenomena noted in lipoid metabolism. Why ketosis develops in one case of diabetes and lipemia in another, and why lipemia retinalis develops in so few cases of the latter, are unsettled questions. Lipemia develops in less than 7 per cent of diabetic persons, as noted at the New England Deaconess Hospital in 1928.[1] McCann found only two cases of lipemia retinalis among one hundred and fifty cases of lipemia.

Although it is known that lipemia will occur in a great number of conditions, such as alcoholism, starvation, asphyxia and phosphorus poisoning (Moore), diabetes is the only disease in which it is frequent enough to be of special significance (Parker and Cullen, and McKee and Rabinowitsch). Lipemia retinalis has been invariably associated with lipemia due to diabetes with one exception, that of a case of leukemia (Wagener).

In discussing the problem, we shall first attempt to explain the phenomena of lipemia as noted by many competent investigators of the subject. Lipids are the substances closely connected with fat metabolism and are normally present in the blood as: (*a*) fat, triglycerides of the higher fatty acids (oleic, stearic and palmitic); (*b*) phosphatides,

1. Joslin, E.: Treatment of Diabetes Mellitus, ed. 4, Philadelphia, Lea & Febiger, 1928.

which are also triglycerides of higher fatty acids with phosphorus in the molecule, namely cephalin and lecithin; (*c*) cholesterol, which is present as pure cholesterol and cholesterol esters of fatty acids. The total fatty acid content of the blood constitutes about 50 per cent of the total blood lipids, the latter varying from 0.5 to 0.8 per cent.[1]

Lipemia is a cloudiness or milkiness of the plasma, dependent on the presence of fat in a particular physical state, namely, an emulsion. On microscopic examination of the blood serum one will find myriads of refractile granules, some of which take an osmic acid stain while others stain with scharlach R (Turney and Dudgeon).

Lipemia does not appear normally in the postabsorptive period, although the blood plasma may contain an excess of cholesterol, lecithin and probably some fat. The ingestion or mobilization of an excessive amount of fat produces a milkiness of blood plasma, lasting a few hours or, under certain conditions, for a considerably longer period. Bloor[2] believes that both the exogenous and the endogenous supply of fat may be the sources of lipemia, but the former is usually more important. He believes that in diabetes there is a deficiency of a hormone, the function of which is to aid in the removal of fat from the blood for storage in the tissues. Allen, however, believes that in diabetes there is a general cachectic condition of the whole organism which affects all the cellular functions, including permeability.[3] It thus prevents the passage of fat into the body tissues, and allows its accumulation in the blood. Another theory advanced for the increased quantity of blood fats is that of an attempt to mobilize an excessive amount of fat for caloric requirements, when the patient loses the power to oxidize carbohydrates, as in diabetes (Muskat). The young diabetic patient, especially, is unable to utilize this fat. This theory also helps to explain the rapid loss of weight at the onset of diabetes.

The mere increase of the quantity of blood fat is not sufficient to cause lipemia, as shown by Wishart in animal experimentation.[4] In McCann's case lipemia was present when the blood contained 9.5 per cent of lipoids, but disappeared at a higher percentage (10.8 per cent). Thus we see that the presence of lipemia depends not only on high blood lipoids, but also on the physical status of the fat, namely, an emulsified state. The cause that brings about this chemical and physical state is as yet unknown, but the presence of acidosis is an important factor (Parker and Cullen).

The retinal changes which are usually associated with diabetes, namely, arteriosclerosis, are found in older diabetic persons. Most of

2. Bloor, W. R.: J. Biol. Chem. **49**:201, 1921.

3. Allen, F. M.: J. Metab. Research **2**:219, 1922; Am. J. M. Sc. **153**:313, 1917.

4. Wishart, M. B.: J. Metab. Research **2**:199, 1922.

the latter have arteriosclerosis and hypertension in contradistinction to lipemia retinalis, which occurs in younger patients.[5] The average age is 25, with extremes of 9 years and 50 years of age. The younger the patient the lower is the power of fat utilization, and thus he is more prone to lipemia and acidosis.

As yet no adequate theory has been advanced to explain the pathogenesis of lipemia retinalis. The mere presence of lipemia is not sufficient. In a series of one hundred and fifty cases McCann noted lipemia retinalis in only two. It has been repeatedly shown on postmortem examination that there are no pathologic changes in the eyes so affected (Fraser, Reis, Heine, Hardy, Muskat). A film of fatty lymph is observed in the perivascular spaces or fat deposits in the adventitia, so that light is abnormally reflected or refracted (McCann). Another explanation offered is that the milky color of the vessels is due to the red blood cells congregating in the middle of the lumen, so that there is an area at the edges of the lumen which is opaque (Cohen).

The ophthalmoscopic findings in this condition have been varyingly described by different observers, depending on the intensity of the lipemia retinalis. They all described the arteries and veins as varying from pale salmon pink to a creamlike color. The peripheral vessels are always lighter in color. The arteries and veins are differentiated with difficulty, the latter being slightly purplish; both arteries and veins are flat, ribbon-like and enlarged in diameter. The central light reflexes are absent or unusually wide. The optic papillae are normal in appearance, while the background of the fundus may be normal or slightly paler, owing to changes in color in the choroidal circulation.

In reviewing the cases, the first changes were noted in the fine terminal vessels, followed later by changes in the larger vessels. In most advanced cases the choroid also changed in color. However, Cohen believes that the choroidal vessels do not change. Because the rate of the circulatory flow is too slow, it does not allow separation of red blood cells and plasma.

Different authors quote different figures for the degree of lipemia necessary to produce lipemia retinalis. This may be due, as stated by Parker and Cullen, to the multiplicity of methods of lipoid determination used, or it may actually have no relation to the degree of lipemia, as has been noted by McKee and Rabinowitsch.

The prognosis in diabetic cases with lipemia retinalis does not differ from that in any other case of impending coma. The high mortality recorded in this condition prior to the use of insulin has been completely reversed since the introduction of this therapy.

5. Wagener, H. P., and Wilder, R. M.: The Retinitis of Diabetes Mellitus, J. A. M. A. **76**:515 (Feb. 19) 1921.

The treatment advocated for both ketosis and lipemia with lipemia retinalis is identical, namely, the administration of large quantities of carbohydrates and insulin in such amounts as may be necessary to clear up the acidosis.

REPORT OF A CASE

History.—L. H., a white man, 30 years of age, was admitted to the medical division of the Morrisania City Hospital on June 4, 1932. His chief complaints were the loss of 30 pounds (13.6 Kg.) within one year and polyuria and polydipsia of the same duration. He had been under treatment for diabetes mellitus on a diet of low carbohydrate and high fat content, with occasional injections of insulin. His condition improved until two months before admission, when he noticed progressive weakness. Until the onset of diabetes, he was subject to frequent colds, but he never had hemoptysis, night sweats or fever. Venereal disease was denied. The family history was irrelevant.

Physical Examination.—The patient appeared weak, but showed no respiratory embarrassment, no acetone odor to the breath and no visual disturbances. The eyes reacted readily to light and in accommodation; there was no muscular imbalance. Ophthalmoscopic examination revealed clear medial and retinal arteries and veins of a pale salmon color. Both vessels were slightly wider and could be differentiated from each other with difficulty. The veins were faintly purplish and wider than the arteries. The vessels appeared flat with no light reflexes. The optic nerves were normal in appearance, though perhaps slightly pale. The choroid was normal.

Pulmonary Examination.—The right upper lung posteriorly revealed inconstant subcrepitant râles. There were no other pathologic findings on physical examination.

Laboratory Observations.—On admission, the urine showed: sugar, ++++; acetone, ++++, and diacetic acid, ++++.

Blood taken for chemical analysis appeared like a mixture of cream and strawberries; on standing, a cream layer, amounting to more than two thirds of the specimen, separated on the top. Chemical analysis of this specimen showed a blood sugar of 226 mg. per hundred cubic centimeters of blood and a carbon dioxide-combining power of 32 per cent. Fat determination was not done on this specimen. A blood count was unsuccessful, owing to the interference of fat globules. The Wassermann reaction of the blood was anticomplementary.

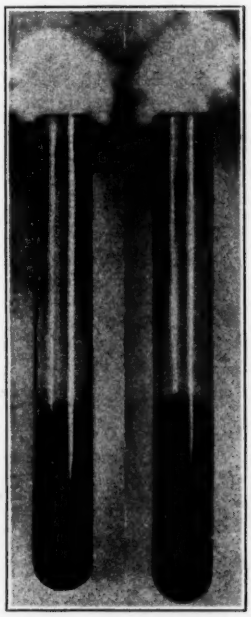
Blood was taken two days later, after vigorous antidiabetic coma therapy with huge doses of dextrose and insulin; the urine was acetonic and free from diacetic acid; a small amount of sugar was still present. The blood sugar amounted to 270 mg. per hundred cubic centimeters. The carbon dioxide-combining power was 48.6 per cent; the total cholesterol, 1,025 mg. per hundred cubic centimeters, and the total lipoid content, 21.7 per cent.

At this time the retinal vessels were still of a light salmon color, but distinctly darker than at the first examination. The veins could be readily differentiated from the arteries.

The patient was given a diet of a high carbohydrate and low fat content (carbohydrates, 200 Gm., protein, 70 Gm., and fat, 38 Gm.), with an adequate amount of insulin to keep him sugar free for one week. During this time the eyegrounds became gradually normal, the larger vessels assuming normal color and caliber first. A specimen of blood on June 13 showed a loss of fatty appearance. The blood sugar measured 195 mg. per hundred centimeters; the carbon dioxide-combining power, 51.2 per cent; the cholesterol, 750 mg., and the total lipoids, 16.3 per cent.

An attempt was made to bring back the former state of lipemia retinalis by giving the patient a diet of a high fat and low carbohydrate content (carbohydates, 30 Gm., fat, 187 Gm., and protein, 70 Gm.) with an adequate amount of insulin, the dosage of which was gradually decreased until none was given. Edema of the ankles began to develop, but subsided after a week. This diet was maintained for seventeen days without a return of lipemia retinalis in spite of the following observations: in the urine, sugar ++++ and acetone ++; blood sugar, 277 mg. per hundred cubic centimeters; total cholesterol, 555 mg.; total fat, 9.3 per cent, and carbon dioxide-combining power, 50.5 per cent.

In view of the pulmonary condition (roentgenograms showed early cavitation of the right upper lung), we hesitated to go any further with our experimental attempt to reproduce the ocular signs, and discharged the patient prescribing a maintenance diet and an adequate amount of insulin.



Right, appearance of blood on the day of admission; left, appearance nine days later.

Roentgenologic examination of the chest showed a chronic tuberculous process involving the right upper lung with fibrosis and early cavitation in the first interspace. The Wassermann test of the blood was negative. The temperature was normal throughout the patient's stay in the hospital.

Comment.—This young diabetic patient with lipemia retinalis began to show changes in the fundus oculi toward normal after twenty-four hours of vigorous antidiabetic coma therapy. Unfortunately a determination of the blood fat was made only on the third day after admission, when the visual degree of lipemia was somewhat less. The total blood lipoids amounted to 21.7 per cent. On a diet of a high carbohydrate and low fat content and adequate insulin the lipemia retinalis

disappeared rapidly. The fundus was normal when the total blood lipoids were 16.3 per cent. The blood, however, still had a slight creamy color.

An attempt was made to reproduce the original lipemia retinalis by means of a diet of a high fat and low carbohydrate content and no insulin for several days. The blood lipoids, instead of rising, fell to 9.3 per cent. This phenomenon was observed repeatedly by Newburgh and Marsh.[6] They attributed it to the ability of the body to utilize the carbohydrate and, as a result, to deposition of fat in the body depots. Parker and Cullen carried their experiment one point further, inducing acidosis. With the onset of acidosis the lipemia retinalis returned. We, however, did not wish to subject our patient to excessive acidosis on account of the complicating pulmonary tuberculosis.

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INSURANCE VALUE OF ONE OR BOTH EYES

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Although the insurance value of one or both eyes has been dealt with by many ophthalmologists, notably by the late Professor Fuchs of Vienna, there are many points which are only imperfectly understood by members of the legal fraternity both in Great Britain and in the Colonies. In England and, I believe, in some of the Colonies, it has been decided by law (and in my opinion most unjustly) that the compensation allowed for the loss of one eye shall be assessed at £375, and for the loss of both eyes at only twice as much, or £750! The Prudential Assurance Society offers £500 for one eye, or limb, and £1,000 for the loss of both eyes or both limbs.

Now from an ophthalmologist's point of view, these amounts are absurd. In many employments, for example, that of a clerk or an employee in a shop, the loss of one eye diminishes the person's working power by only 10 or 15 per cent at the most, so that if the compensation is based on a workman's wages, say, at the rate of £3 a week or £150 a year, he obtains, on the 10 per cent basis, £15 as compensation and, on the 15 per cent basis, only £22, 10s. instead of £375, which the law entitles him to. Furthermore, the law makes no distinction between the various values relating to various kinds of employment. Thus in ordinary office work, such as bookkeeping, typewriting or serving behind the counter, the employee will not be dismissed after losing his eye, whereas in dangerous employments such as mining, blasting and various trades, such as knife grinding, glass cutting, etc., the unfortunate person will be discharged owing to the possible risk of losing the second eye, thus demanding further compensation.

Now the loss of both eyes reduces a workman's capacity to earn his livelihood to nil; the unfortunate man can do nothing except follow some sort of trade like basket-making, or the trades which the teachers at St. Dunstan's home enable him to acquire. But for persons who have lost only one eye the value is enormously large when compared with that allowed for the two eyes, which is absurdly small. As I have already pointed out, the amount assessed by law for the loss of one eye is £375 and for the loss of both, only double that, £750! An annuity is generally valued at half the amount earned in a week or a year. For instance, if, as in the case just quoted, the man were to lose both his eyes, and he was earning £3 a week, or £150 a year, he ought to receive £75 a year or £1, 10s. a week for life. Or if the workman preferred it, he should get a lump sum of £75 multipled by the difference between his age at the time of the accident and 70 years, which is the hypothetic expec-

tancy of life. For example, if the man was 20 years of age at the time of the accident, he should be entitled to either a pension of £75 a year for the rest of his life or a lump sum of £75 multiplied by 50, i. e., £3,750, instead of £750, or by the rules of the Prudential Assurance Company £1,000.

There is another point which has hitherto been entirely overlooked. Does it make any difference as to which eye a man loses? Every one will admit that it makes a vast difference whether a person loses his right or his left arm. If he happens to be right-handed, as most people are, he would be of no use as a clerk for two or three years, and indeed in most trades the loss of the right hand or arm would greatly incapacitate him, whereas the loss of the left arm or hand might make very little difference. Now with regard to the eye, in my opinion the left eye has a slight but definite advantage over the right eye for clerical or reading purposes. This is due to the fact that the blind spot, where the optic nerve enters the eye, lies about 2.5 mm. to the inner side of the macula. Consequently it is important that a person should be able to see a little to the right-hand side of what he is writing or reading, whereas it makes no difference as to the left-hand side, as he has already finished with that part of the sentence. Any one who performs the experiment for himself will notice that when he closes his left eye the point of the pen becomes momentarily invisible as the right eye jumps from word to word, whereas if he closes his right eye no such obscuration occurs. I value the advantage in favor of the left eye at about 5 per cent, but that is merely guesswork, as I have no statistics to rely on. Of course, these statements refer to European languages only, for most eastern languages are written from right to left. I refer to the Hebrew, Arabic, Turkish and Persian languages. In the case of the Chinese and Japanese languages there would be no difference whatever, as they are written from above downward.

Again, in the case of gamekeepers and soldiers who shoot from the right shoulder, the gun or rifle would have to have the stock specially made to fit the right shoulder when using the left eye. Many marksmen declare that the proper way to shoot is to keep both eyes open, and should the double image on the near sight confuse the marksman, he should fix a metal diaphragm or shutter on to the stock which will cut off the right-hand image of the near sight which the left eye receives, and keep both eyes fixed on the distant mark.

In work requiring stereoscopic vision, of course, the loss of either eye would entirely incapacitate a person, but as such occupations are extremely rare they need not be considered here.

From what I have already said, I feel sure that the reader will agree with me that the whole subject of compensation for loss of vision needs careful revision, especially the law as it stands at present.

SIGNIFICANCE OF SPECIFIC INFILTRATION AT THE SITE OF INJURY IN SYMPATHETIC OPHTHALMIA

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The scientific study of sympathetic ophthalmia began with the masterly description of William Mackenzie, in 1835. The second notable event in its history occurred in 1851, with the advocacy, by Augustin Pichard, of the excision of the injured eye as a means of preventing an outbreak of inflammation in the fellow eye. A tremendous stride was the demonstration, by Ernst Fuchs, in 1905, from a painstaking study of anatomic preparations, that sympathetic ophthalmia is a disease entity, distinct from any other malady of the organs of vision. The revelations of Fuchs gave great momentum to the problem of discovering the exciting cause. As the disease was always associated with openings in the eyeball, it was taken for granted that it was of an infectious nature, caused by some micro-organism introduced from without at the place of injury. The histologic observations of Fuchs strengthened this view.

It was natural to draw an analogy between a disease of the eyes following in the wake of perforations and the morbid processes associated with open wounds elsewhere on the surface of the body. As it was discovered that bacteria may gain access to the body by way of open wounds, it was believed that sympathetic ophthalmia was the reaction of the uveal tissues to the presence of virus introduced through an opening in the eye. The bacteriologic world settled down to discover the exciting micro-organism. To this day every effort has failed utterly. In recent times, attempts have been made to explain the phenomenon of sympathetic ophthalmia on the theory that it is due to anaphylaxis or allergy, brought about by the absorption of substances produced in the injured eye by destruction of uveal pigment. This theory has met with great opposition, but nevertheless it has served to throw a shadow over the bacterial hypothesis. Some authorities, in holding to the theory of infection, believe that the responsible bacterium is already present in the body, and that the disease is therefore one of endogenous origin, in which the bacteria localize in the uvea of each eye. The few cases that

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have been reported in which sympathetic ophthalmia was said to have occurred after necrotic sarcomas have added to the confusion of the study of its etiology. There is no escape from the fact that it is a disease that manifests itself after the globe has been opened, and that it rarely occurs—and the rarity should be emphasized—under any other circumstances.

A great number of ophthalmologists still adhere tenaciously to the theory of the exogenous bacterial origin of sympathetic ophthalmia. Since the time that Fuchs wrote his classic papers and Meller pointed out the differences between sympathetic ophthalmia and tuberculosis—twenty years and more ago—no elaborate paper from an anatomic point of view has reached the literature. And yet it is from the study of the pathologic anatomy of the eye that most of the precise information that exists today has been obtained. Every eye removed on account of sympathetic ophthalmia should be subjected to a most careful microscopic scrutiny, in the hope that, from the anatomy, additional knowledge regarding this baffling disease may be gained. There are problems in its microscopy that have not been studied thoroughly.

The present paper concerns itself particularly with the condition existing at the site of the injury. My chief aim is to ascertain in what respects an analysis of the histologic observations there, in abundant material, may speak in favor of or against the theory that sympathetic ophthalmia is the result of a primary infection. No exhaustive survey of the behavior of the uveal tissues in the location of the wound seems ever to have been made, although Redslob,[1] Fliri [2] and Marchesani [3] discussed the topic at some length, in a limited number of cases. As the subject is bound up with that of wound infections in general, it is essential, first, that one have in mind a clear conception of what is meant by the term infection.

GENERAL CONSIDERATION OF INFECTION

Infection is defined as the invasion of the body by any microorganism foreign to it. In some instances the bacteria pass through the portal of entry without lodging there, going directly to other parts, as does Plasmodium malariae. In other instances they are retained at the portal of entry, as in diphtheria, in which the bacillus remains at the place of entrance and produces its effects by the elaboration of

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intense toxins. In still another group, the bacteria may be partly retained at the portal of entry and partly carried off by the circulating lymph or the blood. In the lymph tract the bacteria are arrested in the lymph nodes, whereas from the blood stream they may be deposited in any organ of the body. Coming under this head are tuberculosis, syphilis and wound infections. As a direct result of the retention of bacteria at the portal of entry there is set up a biochemical reaction that manifests itself by a local accumulation of cells. This focus is spoken of as a primary lesion. It is possible, therefore, for a primary infection to occur with or without a primary lesion. In some cases of infection

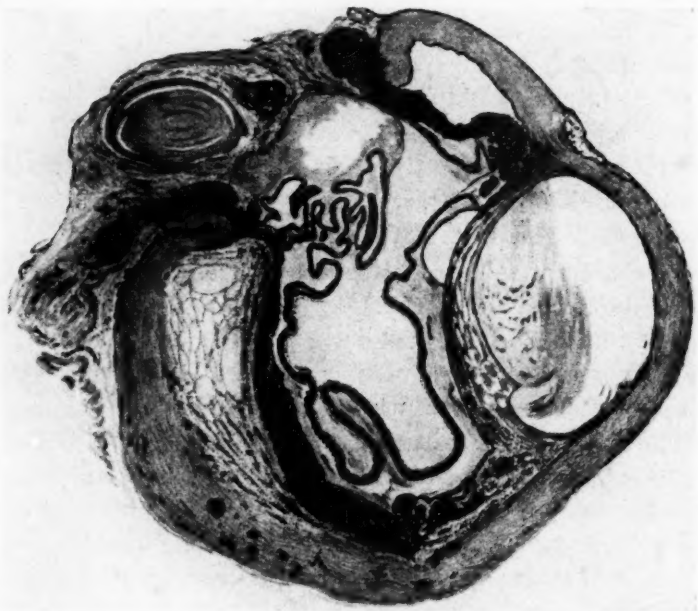

Fig. 1.—Iris: massive infiltration in prolapse. Ciliary body: massive infiltration in prolapse. Choroid: heavy infiltration on the side of the injury; almost none elsewhere. Changes in favor of a primary lesion: infiltration of prolapse greater than elsewhere; infiltration of the choroid, greater on the side of perforation; elsewhere almost free; emissaria infiltrated on the side of the injury; free elsewhere. Changes against a primary lesion: none.

the primary lesion is so small or so concealed that it escapes notice. It has been demonstrated that in the scars of primary lesions, as in old cicatrices of carbuncles or healed-over alveolar abscesses after the extraction of teeth, virulent micro-organisms may be found for an indefinite period. Furthermore, it has been shown that infection may take place through an intact and healthy mucous membrane. Calmette, by merely instilling tubercle bacilli of a virulent strain in the conjunctiva of guinea-pigs, produced generalized tuberculosis without any demonstrable primary lesion or involvement of the eye.

DEFINITION OF SYMPATHETIC UVEITIS

The bilateral disease that may come on after an eyeball has been opened by injury, operation or perforating ulcer consists anatomically of an infiltration of the uveal tract, composed of lymphocytes and epithelioid cells, with or without giant cells. The infiltration in the uninjured eye is similar in every respect to that in the injured eye. In the uninjured eye the picture is a pure one, free from any sequelae of an injury. No other disease of the eye presents the same degree of uniformity in its histologic aspects as does sympathetic ophthalmia. The identical elements

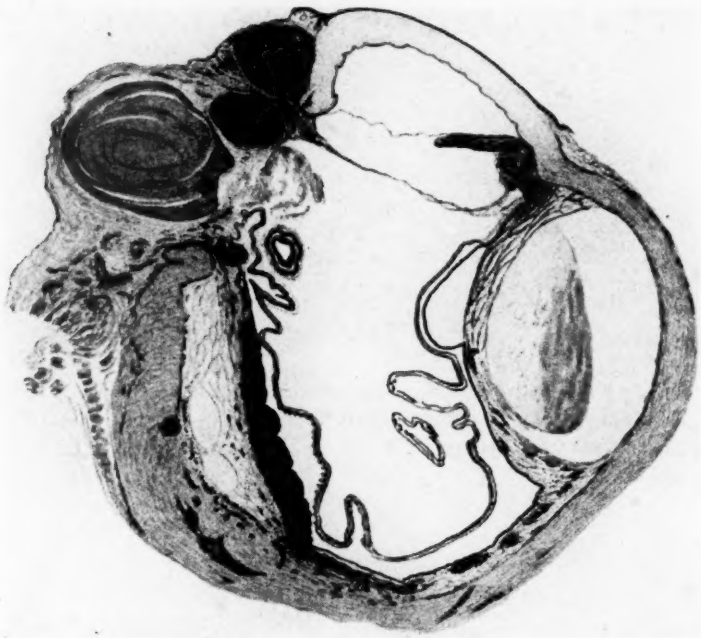

Fig. 2.—Another view of figure 1, showing relatively slight movement of the iris and ciliary body opposite the wound. Note that the infiltration of the choroid appears to be independent of that anteriorly. That the entire process is continuous is demonstrated in figure 1.

are always present in the infiltration of the uveal tissue, collected in nodules or diffusely disposed. It is only after careful examination that a nodule may by chance be found in the retina.

GENERAL SURVEY OF THE MATERIAL

The material employed in this study of the condition of the uvea at the site of the wound comprised microscopic sections of one hundred and one eyes in which the specific infiltration of sympathetic ophthalmia was found. In the consideration of many of the eyes there were

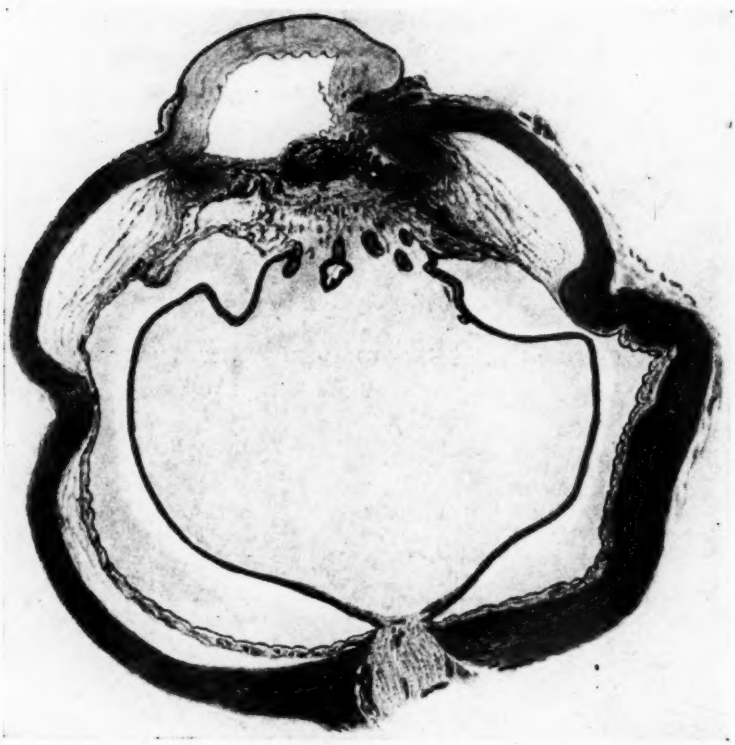

Fig. 3.—Iris: epithelioid cells in the prolapse, and nowhere else. Ciliary body: nodules of lymphocytes. Choroid: scanty foci of lymphocytes. Changes in favor of a primary lesion: epithelioid foci only in incarcerated iris. Changes against a primary lesion: none

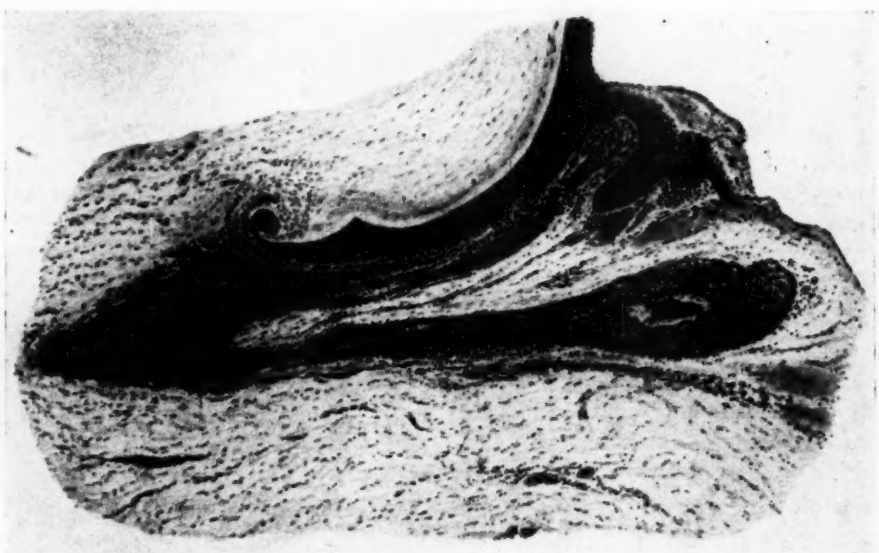

Fig. 4.—Detail of figure 3, showing foci of epithelioid cells and remnants of the structure of the iris.

available only one or two slides, whereas in a few cases many were to be had. The following general observations were made:

1. *Interval Between the Time of Injury and Enucleation.*—In seventy cases the period that elapsed from the date of the injury to the date of enucleation covered the wide range of from three weeks to forty-one years. In thirty-one cases the date of the injury was not

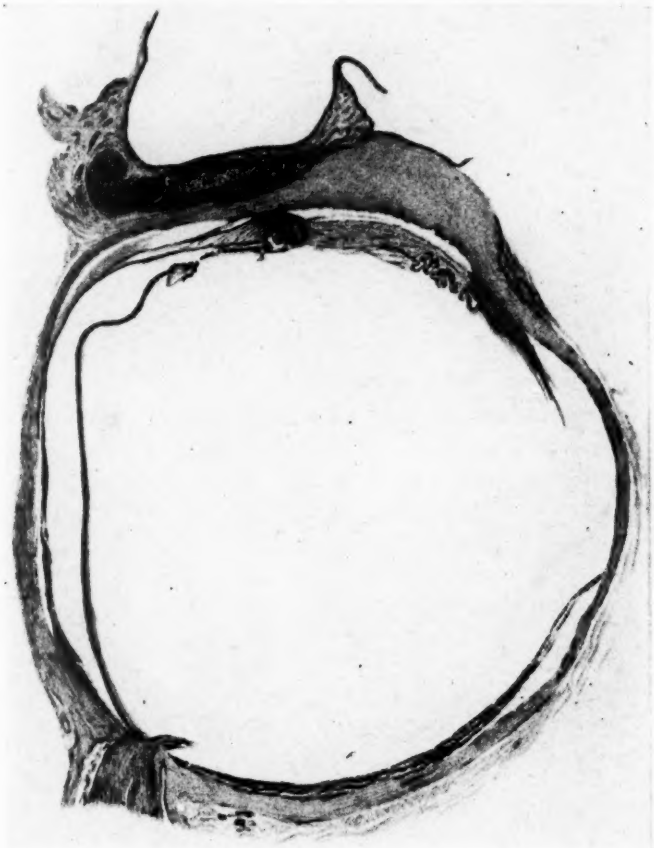

Fig. 5.—Iris: massive infiltration of the prolapse. Ciliary body: massive infiltration on the side of the injury, vis-à-vis little infiltration. Choroid: many typical nodules, diffuse lymphocytic infiltration; ora serrata free. Changes in favor of a primary lesion: massive infiltration of prolapsed iris. Changes against a primary lesion: diffuse infiltration of the choroid; ora serrata free.

stated. In the majority of the cases in which the date of the injury was given, the eyes were removed, within the first two or three months after the injury.

2. *Interval Between Outbreak of Inflammation in the Uninjured Eye and Enucleation.*—In fifty-eight of the histories there were data covering this point. The fellow eye became inflamed in twenty-four

cases in from one to fourteen days before the enucleation. In the remaining thirty-four cases the interval ranged from fourteen days to six and one-half years.

These figures may not represent the actual duration of the disease in the uninjured eye before the time of enucleation, but refer to the time of its detection. The inflammation may in certain instances have existed a number of days before it was discovered. It is well known

Fig. 6.—Iris: massive infiltration of prolapse. Ciliary body: heavy typical infiltration, more on the side of the wound than opposite. Choroid: large typical field of infiltration in the posterior segment opposite the rupture. Changes in favor of primary lesion: massive infiltration of prolapse. Changes against primary infection: a large field of infiltration in the choroid opposite the wound.

clinically that cases are encountered in which the second eye shows precipitates on Descemet's membrane and neuroretinitis, in spite of which the patient may be ignorant of any involvement of this eye. Many of the cases in the collection date back to the days prior to the invention of the slit-lamp, the greatest merit of which is the aid that it renders in the detection of sympathetic ophthalmia.

3. *Interval Between Enucleation and Outbreak of Inflammation in the Fellow Eye.*—There were ten cases in which the fellow eye became inflamed after the removal of the injured eye, as follows:

No. of cases	1	3	1	1	1	1	2
No. of days after enucleation	40	18	9	8	6	4	Unknown

4. *Specific Infiltration in the Injured Eye, With No Signs of Inflammation in the Fellow Eye.*—There were seven such cases:

No. of cases	1	1	1	1	1	1	1
Interval between injury and enucleation, days	2	4	8	18	40	A few	Unknown

HISTOLOGIC EXAMINATION OF THE MATERIAL

1. *Varying Amount of Specific Infiltration.*—The infiltration was found to be heavy in seventy-one cases and moderate or scanty in thirty. In three cases the infiltration was so massive as almost to fill the globe. An infiltration of limited extent, with few exceptions, characterized those eyes that were removed a few days after the outbreak in the fellow eye. Even less infiltration was noted in the ten cases in which the infiltration broke out in the other eye after the enucleation of the injured eye. This also held good for the seven cases in which there was no clinical evidence of involvement of the fellow eye. According to the histories, it appears that a massive infiltration may develop within three or four weeks after the injury, as was observed by Meller.

2. *Specific Infiltration Complicated by Endophthalmitis.*—Relatively few of the one hundred and one eyes examined gave evidence of having suffered from septic endophthalmitis. This supports the observation of other writers that sympathetic ophthalmia most often runs its course uncomplicated by the presence of pus-producing bacteria.

DIFFICULTIES OF MICROSCOPIC STUDY OF SYMPATHETIC INFILTRATION AT OR NEAR SITE OF INJURY

Often in typical cases of specific infiltration there are pitfalls to be avoided. Thus, in the iris newly proliferated fibers of the dilatator muscle, formed under the stimulation of toxins, closely resemble epithelioid cells containing pigment, and are misleading. Cells derived from the dilatator muscle are more sharply defined and take a deeper stain than typical epithelioid cells. It is not unusual for the endothelium of a small vessel, when undergoing proliferation, in cross-section, to simulate a giant cell. Isolated giant cells in the vicinity of the wound should be carefully examined lest they prove to be foreign body giant cells, embracing fragments of lens capsule or particles of lens matter.

Fig. 7.—Detail of figure 6, showing the massive infiltration of the prolapsed iris. The process is in an advanced stage, as is indicated by large foci of epithelioid cells.

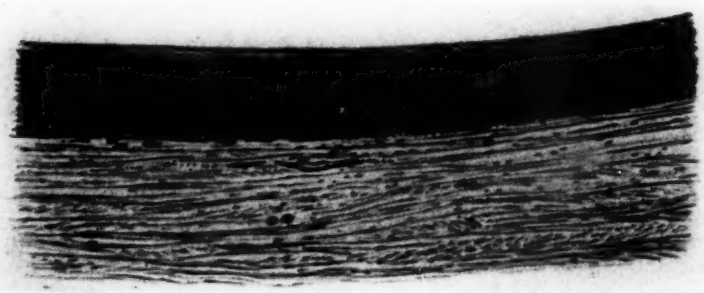

Fig. 8.—Detail of figure 6. Epithelioid cells are present in the choroid, but the process is not so far advanced as that in the prolapsed iris.

The liquefaction that is sometimes seen in streaks in large masses of specific infiltration is not to be confounded with the caseation of tuberculosis. The correct interpretation of many points is a matter of pathologic knowledge and experience.

SITE OF THE INJURY

In eighty cases the opening was found to be in the anterior segment of the globe. In one case the injury was a short distance posterior

Fig. 9.—Typical nodules in the ciliary muscle near the wound in a case in which a particle of steel was removed.

to the ciliary body. In twenty cases the specimens did not include the opening in the globe.

CLASSIFICATION OF CASES ACCORDING TO QUANTITY OF INFILTRATION

Group I: Cases with an equable distribution. Of these there were fifty-eight that showed about the same amount of infiltration in the various divisions of the uvea. They formed the largest group.

Group II: Cases showing infiltration at the site of the wound and nowhere else. There were two such examples.

Group III: Cases showing infiltration overwhelmingly greater at the site of the wound than elsewhere. There were nine of these.

Group IV: Cases showing infiltration absent at the site of the wound although present elsewhere. There were two of these.

Group V: Cases showing less infiltration at the site of the wound than elsewhere. Of these there were ten.

Anatomic Findings in Groups.—Group I: That a large number of these cases showed no excessive amount of infiltration anywhere

Fig. 10.—Typical nodules of epithelioid and giant cells in the choroid at the place of perforation in the sclera, in another case in which steel was removed.

was attributed either to a weakness of the virus or to the inability of the uvea to react. In some of the cases great damage had been done to the iris and ciliary body at the moment of the injury. In one case the prolapsed iris was excised shortly after the injury. It often happened that in the region of the wound there were only fragments of uvea left in which infiltration could take place. Probably for these reasons, in a series of cases, there was no great infiltration in the anterior segment. There were specimens in which necrotic areas in the iris near the wound were entirely free from infiltration, whereas the stroma of the iris elsewhere was transformed into infiltration, and at the same time infiltration was found in other parts of the uvea.

Posteriorly in three cases spots of old chorioretinitis stood out free in the midst of large fields of specific infiltration. A certain degree of vitality of the uveal tissue seems necessary for the development of infiltration. In a few cases the sequelae of the traumatism dominated the picture anteriorly, where one found much scar tissue and little specific infiltration. Posteriorly, the specific infiltration progressed, as a rule, unhindered by any damage to this part of the uvea at the time of injury.

Groups II and III: These groups, showing the infiltration confined to the wound or overwhelmingly greater in its neighborhood than elsewhere, permit of a limited interpretation. The local infiltration here is evidently an expression of a wound infection.

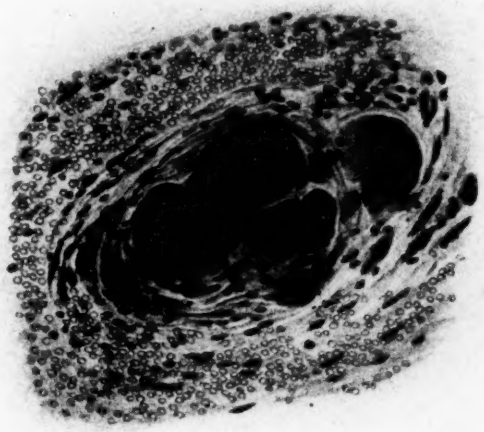

Fig. 11.—Detail in the case shown in figure 10, revealing giant cells on the external surface of the sclera at the point of perforation, embracing threads of the lens capsule.

It was a striking fact that many of the cases of massive tumor-like infiltration in the anterior segment developed after large ruptures or followed cataract operations. Large openings, with widely separated margins and extensive prolapse of the iris and ciliary body, were those that presented the greatest amount of infiltration at the site of the wound. Possibly there would have been a greater number of specimens of massive infiltration but for the fact that severely damaged eyes are generally promptly enucleated.

Groups IV and V: Not too much emphasis should be laid on the two cases in group IV that showed an absence of infiltration at the site of the wound. In each instance only a slide or two were available for study. Serial sections might have revealed infiltration also in the iris. One of the two cases showed typical nodules in the

ciliary body on the side of the wound. In the cases of group V the greater amount of infiltration in the posterior over that in the anterior segment was thought to be due to the fact that the choroid posteriorly is normally thick and very vascular, affording ample stroma for the accumulation of cells. However, a massive tumor-like infiltration of the choroid existed in only one case, in contradistinction to its rather frequent occurrence in the iris.

Separation of Anterior and Posterior Fields of Infiltration by a Free Zone at Equator.—In many cases the infiltration in the iris and

Fig. 12.—Appearance in a weakly "pro" and strongly "contra" case. Iris: massive infiltration. Ciliary body: little infiltration. Choroid: massive infiltration in the posterior segment. Change in favor of a primary lesion: massive infiltration of the prolapsed iris. Changes against a primary infection: choroidal infiltration greater than that of the iris.

ciliary body was separated from that in the posterior part of the choroid by a broad equatorial zone remarkably free from infiltration. It has been suggested that the distribution of the specific infiltration in apparently independent fields is brought about by the introduction of a virus into the eye at different points by way of the general circulation. However, according to the material studied, the infiltration proceeds in a zonular fashion, beginning at the wound and first infiltrating the iris more or less as a whole, then the ciliary body more or less as a

whole and finally breaking out in the posterior part of the choroid, leaving the equatorial region almost free from involvement. This method of infiltration resembles that of tuberculosis in its spread from the iris and ciliary body posteriorly, as tuberculosis often leaves a comparatively free zone at the equator in its passage from the ciliary body to the region around the optic nerve. In both diseases an examination of serial sections will usually reveal that the equatorial region is in reality not entirely free, but that it contains small foci forming links between the two zones. A number of times a direct path of infiltration on the side of the wound could be traced from the incarcerated iris

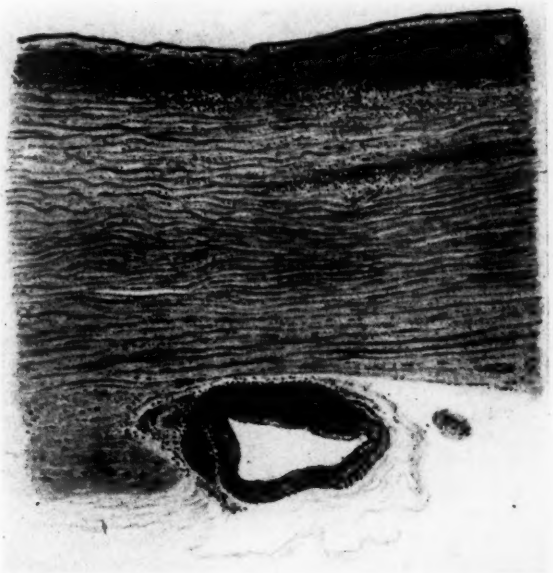

Fig. 13.—Appearance in a "contra" case (after cataract extraction). Iris: mild typical infiltration. Ciliary body: slight infiltration. Choroid: heavy diffuse infiltration as in illustration, with extra-ocular extension along a blood vessel. Changes in favor of a primary lesion: none. Changes against a primary lesion: more infiltration of choroid than of iris, with marked extension, through emissaria, to the external surface of the globe.

backward through the ciliary body and choroid to the margin of the optic nerve. In both sympathetic ophthalmia and tuberculosis, the infiltration at the ora serrata is poorly developed, apparently owing to the thinness of the uvea at this point. In one case, traction at the ora serrata, produced by a detached retina, seemed to have acted as a hindrance to the backward progress of an infiltration in the ciliary body, since no specific nodules were discovered anywhere posterior to this point, whereas the infiltration was heavy anterior to it. A favorite

TABLE 1.—*"Pro" Cases (Cases Showing Signs in Favor of the Theory of Primary Infection)*

Case	Dating from Enucleation: Time of Injury	Dating from Enucleation: Time of Sympathetic Ophthalmia	Age, Years	Kind of Injury	Changes in Iris	Changes in Ciliary Body	Changes in Choroid	Changes Favoring Primary Infection	Changes Against Primary Infection
1	1 mo.	4 days after	9	Scleral rupture from cow's horn	Tumor-shaped infiltration in prolapse	Tumor shaped infiltration in prolapse	Very heavy infiltration on side of injury	Infiltration in prolapse greater than elsewhere; choroid more infiltrated near injury, with invasion of sclera; elsewhere almost free	None
2	4 mos.	?	52	Extraction of luxated lens	Epithelioid cells only in prolapse; elsewhere lymphocytes	Nodules of lymphocytes	Scanty foci of lymphocytes	Epithelioid foci only in prolapse	None
3	?	After ?	?	Scleral rupture; subconjunctival luxation of lens	Tumor-shaped infiltration in prolapsed iris	Tumor-shaped infiltration on side of injury; opposite side only slight infiltration with giant cells	Many typical nodules, diffuse lymphocytic infiltration; ora serrata free	Tumor-shaped infiltration of iris	Diffuse choroidal infiltration (ora serrata free)
4	6 wks.	2 weeks before	8	Corneal wound from scissors	Tumor-shaped infiltration in prolapsed iris	Tumor-shaped infiltration with giant cells	Diffuse tumor-shaped infiltration	Iris and ciliary body more infiltrated than choroid	Diffuse choroidal infiltration; specific optic nerve infiltration
5	Some time	?	?	Corneal wound	Tumor-shaped infiltration	Heavy, typical infiltration	Scanty nodules	Iris more infiltrated than ciliary body and this more than choroid	Diffuse choroidal infiltration; ciliary body opposite site of wound heavily infiltrated

6	15 mos.	1 month before	58	Operations for glaucoma; cyclodialysis (twice); LaGrange operation posterior sclerotomy	Atrophic, free	Epithelioid cells and giant cells in flat part	Heavy lymphocytic infiltration and typical nodules	Tumor-shaped infiltration in prolapsed iris	Large focus in choroid opposite site of rupture
7	5 wks.	?	9	Scleral rupture from blow with spoon	Tumor-shaped infiltration in prolapsed iris	Heavy typical infiltration mostly on side of wound	Typical, heavy focus in posterior segment opposite rupture	Tumor-shaped infiltration in prolapsed iris	Large field in choroid opposite rupture
8	3 mos.	?	68	Cataract extraction with prolapse of iris	Tumor-shaped infiltration and giant cells in prolapsed iris	Heavy typical infiltration	Heavy infiltration and typical nodules; ora serrata but slightly affected	Iris more than ciliary body, this more than choroid	Diffuse choroidal infiltration; iris opposite wound is tumor-shaped
9	6 wks.	?	18	Splinter of steel and extraction	Everywhere typical nodules	Heavy typical infiltration everywhere	Heavy infiltration; nodules in muscle near wound	Especially heavy infiltration on side of wound; nodules in muscle near wound	Infiltration of iris and ciliary body opposite sheath; diffuse choroidal infiltration
10	6 wks.	?	68	Glaucoma iridectomy	Tumor-shaped infiltration	Heavy typical diffuse infiltration	Heavy diffuse infiltration	Tumor-shaped infiltration of prolapse	Choroiditis
11	Several weeks	?	?	Injury from cow's horn	Very little	Tumor-shaped infiltration near rupture; heavy infiltration opposite	Heavy diffuse typical infiltration on side of wound, posterior more than anterior	Greatest infiltration on side of wound	Infiltration of ciliary body greater than that of iris; that of choroid greater posteriorly than anteriorly

site for well developed nodules in sympathetic ophthalmia and in tuberculosis is the root of the iris.

The equatorial region of the choroid is largely one of veins, which here run straight and direct to empty into the vortices. It is possible that in the fluids of these parts the virus of sympathetic ophthalmia is carried along more rapidly than elsewhere and so escapes localization. It is certain that from the choroid the infiltration invades the uveal

Fig. 14.—Same eye as that in figure 13, showing involvement of the pial and arachnoid sheaths of the optic nerve.

tissue that lines the walls of the emissaria. The invasion of the emissaria was, as a rule, more marked on the side of the wound than elsewhere.

In severe cases the equatorial region was found infiltrated, with the rest of the uveal tract. The infiltration of this zone seemed to be brought about by a spreading of the anterior and posterior fields until they coalesced. In one case of heavy infiltration the union of the anterior and posterior zones of infiltration was interrupted, at one point at the equator, by pressure exerted against the choroid by a lens

Fig. 15.—Appearance in a strongly "pro" and weakly "contra" case; one of glaucoma after two cyclodialyses and a Lagrange operation. The illustration shows the flat part of the ciliary body heavily infiltrated with epithelioid cells and giant cells in the vicinity of the wound. The iris is atrophic and not infiltrated. Choroid: numerous foci of lymphocytes with a few epithelioid cells. Changes in favor of a primary lesion: infiltration heaviest and in a more advanced phase near the wound; iris free. Evidence against a primary lesion: infiltration in the choroid.

Fig. 16.—Advanced stage of specific infiltration involving the entire uveal tract in continuity. The anterior segment is far more heavily implicated than the posterior part. The process appears older in the anterior part, where there is some breaking down in the infiltration. As the entire uvea is infiltrated, it is difficult to decide where the process began, but the greater quantity and higher degree of development in the region of the wound suggest that this was the site of the primary lesion.

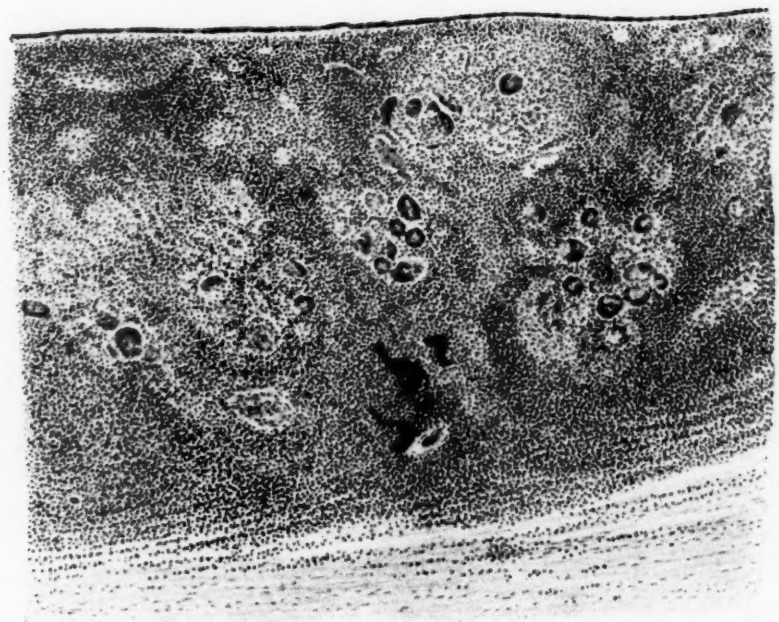

Fig. 17.—Detail of figure 16, near the optic nerve, showing the "complex" of lymphocytes, epithelioid cells, and giant cells. The choriocapillaris is not infiltrated.

that had fallen back into the vitreous and become adherent to the choroid. In another case a similar interruption of the infiltration at the equator was caused by the pressure of a subchoroidal hemorrhage against the choroid.

Age of Specific Infiltration.—It is remarkable that in almost every case, in old injuries as well as in new, the infiltration gave the impression of being recent—of not more than a few weeks' duration. A comparison of the age of the infiltration in the various parts of an eye generally revealed that the infiltration was older in the anterior segment. Here in many cases epithelioid and giant cells could be found, whereas posteriorly the infiltration was in the initial stage, consisting only of nodules of lymphocytes. At other times, the cells anteriorly took the stain poorly, as compared with those in the choroid, indicating regressive changes in the former. Small areas of necrosis were not uncommon in the anterior segment, but only once were they seen in the posterior segment. In two cases, in which the anterior segment was largely walled off by cyclitic membranes, the infiltration within the membranes was highly necrotic, whereas that in the choroid appeared quite fresh.

TABLE 2.—*"Contra" Cases (Cases Showing Signs Against the Theory of Primary Infection)*

No.	Time Previous to Excision	Time of Outbreak of Sympathetic Ophthalmia	Age, Yrs.	Kind of Injury	Changes in Iris	Changes in Ciliary Body	Changes in Choroid	Changes Favoring Primary Infection	Changes Against Primary Infection
1	Unknown	Unknown	75	Cataract extraction	Prolapse free from infiltration	Typical nodules on side of opening	Many typical nodules	Infiltration of ciliary body on side of wound	Iris free; choroid highly infiltrated
2	3½ mos.	1 mo. after excision	59	Struck with piece of iron	Prolapse free from infiltration	Only lymphocytes	Many typical nodules	None	Iris prolapse free; choroid highly infiltrated

CLASSIFICATION OF CASES IN RELATION TO THEORY OF PRIMARY INFECTION

The deciding factor in the classification of each case in relation to the theory of primary infection was the amount of infiltration in the anterior segment of the uvea as compared with that in the posterior segment. According to this method, the eighty-one cases in which microscopic slides were made of sections that went through the wound were divided as follows:

1. "Indefinite" cases. Under this heading were the fifty-eight cases of group I. Because the infiltration was about the same in amount in the

various divisions of the uvea, it was considered that they did not reveal anything in favor of or against the theory of primary infection from the standpoint of quantity.

2. "Pro" cases. The two cases of group II, showing infiltration only at the site of the wound, and the nine cases of group III, showing an overwhelmingly large amount, were interpreted as speaking strongly in favor of the theory of primary infection. The two cases of group II fulfilled every requirement of the definition of a primary lesion, as they showed local accumulations of cells at the site of an open wound.

3. "Contra" cases. The two cases of group IV, because they showed an absence of infiltration at the site of the wound, and the ten cases of group V, because they showed less infiltration there than elsewhere were considered to speak against the theory of primary infection.

In the accompanying tables details are given of the "pro" and "contra" cases.

CONCLUSIONS

1. The outstanding feature of this analysis was the finding of two cases of primary lesions in sympathetic ophthalmia. To these were added nine cases in which the infiltration was overwhelmingly greater in the region of the wound than elsewhere. Similar observations were reported by Redslob and Marchesani, the former of whom was the first to speak of the infiltration at the site of the wound as a primary lesion. Fliri denied the existence of a primary lesion in sympathetic ophthalmia, arguing that what Redslob interpreted to be such were but the signs of a localization of micro-organisms that were already circulating in the blood before the injury took place. To this one may reply that the uveal tissue is often severely damaged in contusions and yet no local specific infiltration develops afterward. The essential thing is that the globe must first be opened. Fliri further argued that if sympathetic ophthalmia is due to a primary infection, one should be able to demonstrate the primary lesion in every case. He did not take into consideration the facts that primary infection may occur without a primary lesion and that a primary lesion may be so insignificant or hidden as to escape notice. The failure to find a primary lesion proves nothing, whereas the discovery of a single primary lesion is of the greatest significance. Moreover, it is recalled here, as suggestive of a primary lesion, that the infiltration in the anterior part of the uvea appeared to be older than that in the posterior part. In certain cases the path of the infiltration from the site of the wound to the choroid could be traced by continuity with as much certainty as that from the choroid to the emissaria.

2. It does not so much matter how few the cases of primary lesions are that have been reported as the fact that they do exist. The establishment of their existence strengthens the original theory, and weakens all others, that sympathetic ophthalmia is caused by a bacterium whose portal of entrance is an opening in the eyeball.

ABSTRACT OF DISCUSSION

Dr. F. H. Verhoeff, Boston: It seems probable that what happens is this: A wound is present, and when the germs reach it, a certain incubation period is required to produce a lesion. Ordinarily about six weeks are required. Then the germs go into the blood and are carried to both eyes at the same time. It seems to me that when just a little inflammation is found at the site of the wound, the involvement of the other eye is not due to continuity, but to the germs which have gotten into the blood and been carried to the choroid of both eyes in the same way. Of course, this is assuming the theory that it is due to a germ, and I believe that very strongly; however, I do not believe that one can accept that as true until the germ is found. Moreover, allergy may play a part in addition, and that may be the factor that makes all these later conditions in the uveal tract.

Dr. Jonas S. Friedenwald, Baltimore: Recently we have been trying to obtain some new criteria to throw light on whether or not the disease was allergic. In going over a series of twenty cases we found that many more than we previously believed showed definite small tubercle-like lesions of the retina. In fact, ten of the twenty patients had such small tubercles at the site of the wound, and in the tubercles were found pigment granules. It then occurred to me that if the character of the reaction of the epithelioid cells was determined to be allergy to pigment, then if one injected uveal pigment into the patient at other sites one should elicit a characteristic reaction.

In normal persons the reaction at the site of injection into the subcutaneous tissues is very slight. In two patients with sympathetic ophthalmia, however, the character of the reaction about the area of skin tested was identical with that seen in the eye: great masses of epithelioid cells extending 2 or 3 mm. away from the site of the pigment. On looking at the tissue under high-power magnification it was almost impossible not to be convinced that it was not a higher-power field of an eye with sympathetic ophthalmia.

Dr. Bernard Samuels: I fully agree with what Dr. Verhoeff said, but that really was not my idea about it. I wanted to bring out particularly that these so-called free zones were not always free. Of course, it looks as if the microorganism was in the wound and attacking the choroid simultaneously.

In regard to Dr. Friedenwald's comment, the situation resolves itself into this: Disease breaks out after the eyeball has been opened. There are other conditions in which the uveal pigment is destroyed, but those conditions are not sympathetic ophthalmia. It seems absolutely essential that the eyeball be opened.

PIGMENTATION OF THE OPTIC NERVE

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This article will deal mainly with the occurrence of uveal pigment, in the form of chromatophores in the lamina cribrosa, as a normal physiologic variation. The establishment of this fact will be considered a reason for anticipating in the disk any manifestations of pigment which are encountered in the uveal tract, and in this connection three cases of melanoma of the disk will be reported. Other forms of pigmentation will be discussed, including those arising from the pigmented epithelium, from hematogenous granules of pigment and from siderosis. Finally, several conditions simulating a pigmented disk will be mentioned.

PIGMENTATION OF THE LAMINA CRIBROSA

The external laminae, or laminae scleralis, are continuations of the scleral elements adjacent to the nerve, while the internal laminae, or laminae choroidalis, are continuations of the choroidal elements by way of the border tissue. These laminae usually contain no pigment cells, but in eyes in which the uveal tract is considerably pigmented chromatophores may be found in them, particularly in the lamina choroidalis. The lamina cribrosa may be considered then as potentially pigment-bearing tissue in which pigment may manifest itself in excessively pigmented persons and races (fig. 1, 2 and 3). It is also a normal finding in the eyes of many animals, including sheep, cows, pigs, camels, crocodiles and alligators.

I have never seen ophthalmoscopically pigmentation of the lamina cribrosa nor have several of my colleagues whose practice is in districts where the Negro element is large. This may be explained by the fact that the peripheral portions of the lamina cribrosa, which are more often and more densely pigmented than the central portions, are hidden from view by the opaque glial tissue constituting the stroma of the nerve fibers of the disk. When the pigment extends to the central portion it should be seen ophthalmoscopically at the base of the physio-

Read before the American Ophthalmological Society, New London, Conn., June 29, 1932.

logic cup, particularly as it occurs chiefly in the lamina choroidalis which is more accessible to view. Ogawa [1] has observed it ophthalmoscopically in normal Japanese eyes.

In view of the fact that the lamina cribrosa is potentially a pigment-bearing tissue, it is not surpising to find, although very rarely, a melanoma of the disk. Three such cases are described here.

Case 1.—A man, Jewish, aged 70, complained of some irrelevant symptoms of the lids. Positive findings consisted of a mild conjunctivitis and a small refractive

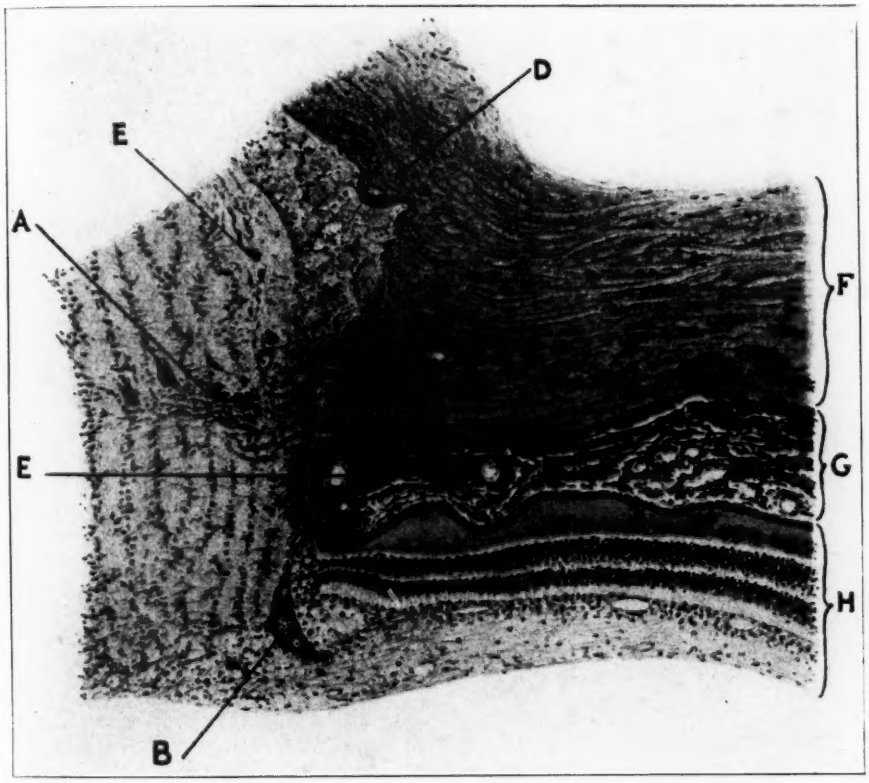

Fig. 1.—The temporal side of the disk and adjacent retina, choroid and sclera to show pigmentation of the lamina cribrosa: *A*, pigmentation of the lamina cribrosa; *B*, distal or retinal portion of pigmented cilioretinal artery; *C*, proximal or choroidal portion of cilioretinal artery; *D*, pigment in the dura; *E*, pigment in the pia; *F*, sclera showing pigmentation lamina fusca and inner lamellae; *G*, choroid, and *H*, retina which is detached, and between it and the pigment epithelium there is fluid in which are floating pigment epithelial cells. (Reese: Am. J. Ophth. **8**:865 [Nov.] 1925.)

error, in addition to a melanoma in the disk of the left eye. He had been examined thirty years previously in Philadelphia, at which time many doctors

1. Ogawa, K.: Ueber Pigmentirung des Sehnerven, Arch. f. Augenh. **52**:437, 1905; Die normale Pigmentirung im Sehnerven der Japaner, ibid. **55**:106, 1906.

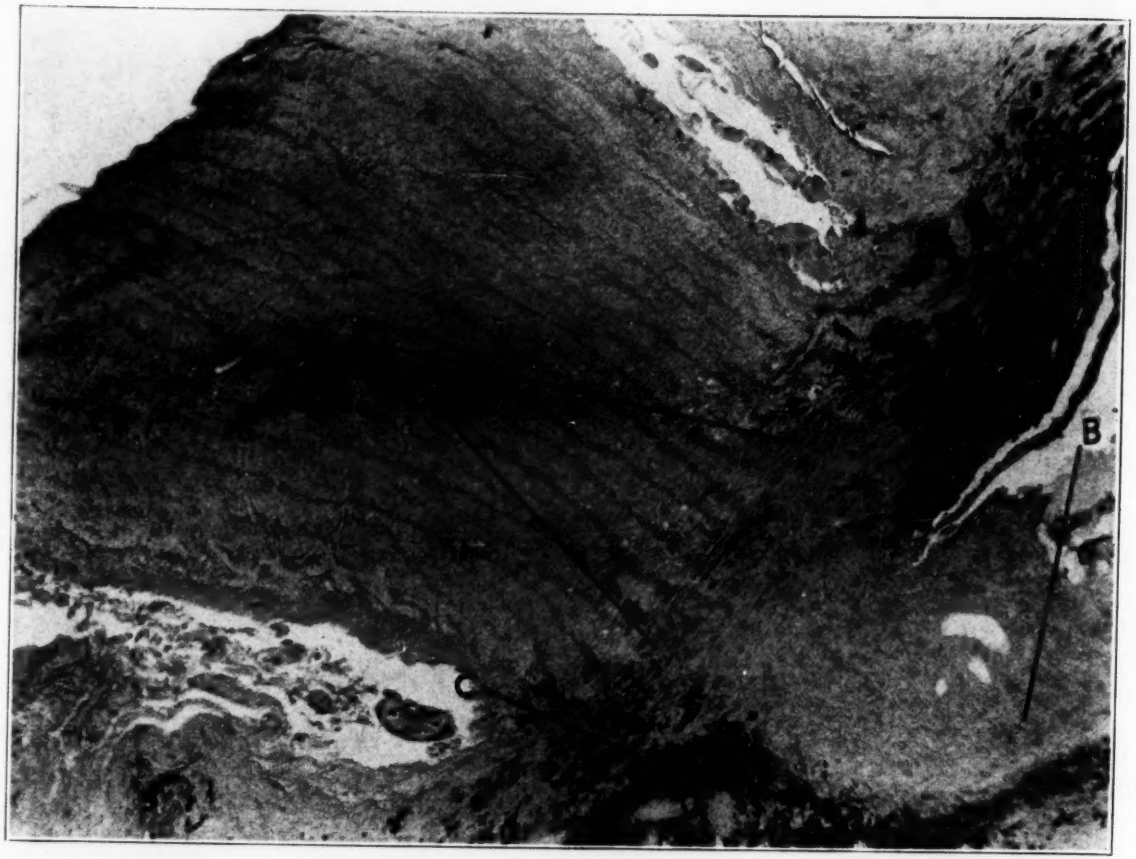

Fig. 2.—Optic nerve to show pigmentation of the lamina cribrosa. *A*, pigmented lamina cribrosa; *B*, detached retina, and *C*, melanosarcoma choroid.

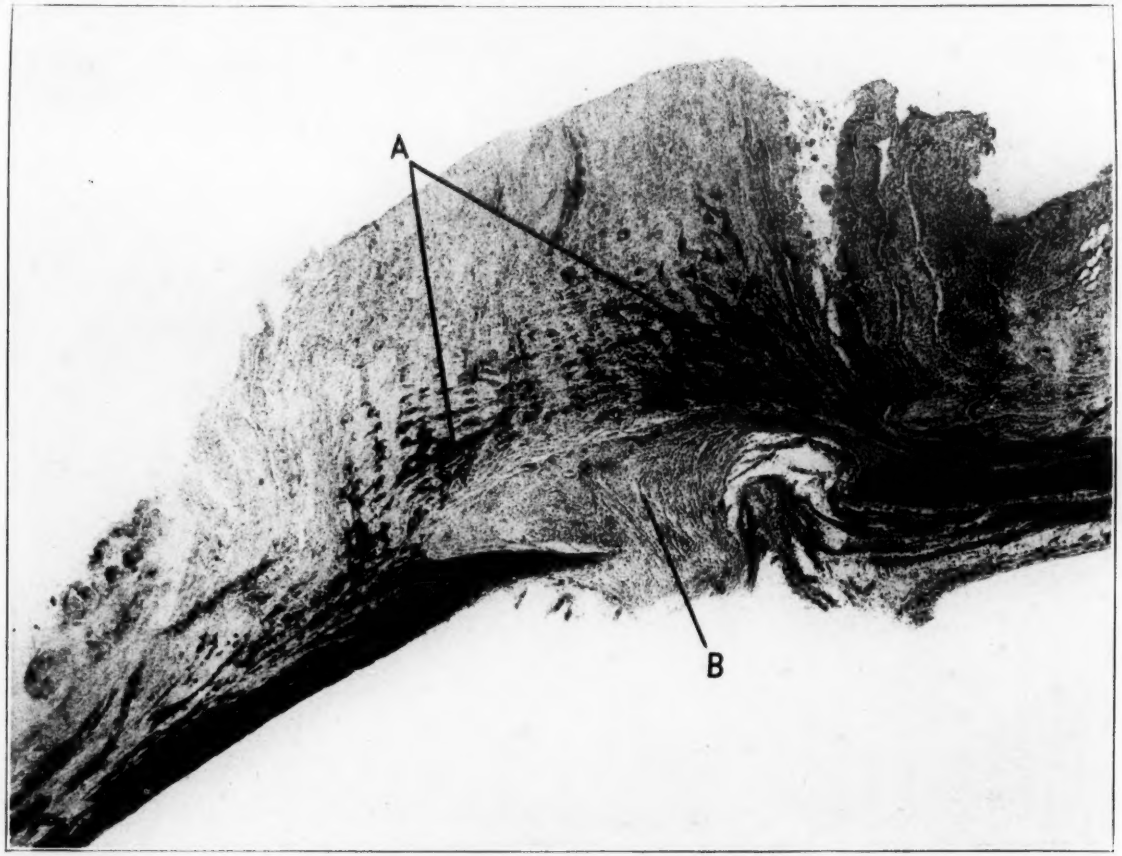

Fig. 3.—Optic nerve to show pigmentation lamina cribrosa: *A*, pigmented lamina cribrosa, and *B*, cupping of the disk partially filled with glial tissue. The retina on the nasal side has been lost in preparation. (Doherty: Am. J. Ophth. **10**:248 [April] 1927.)

looked into his left eye because of some unusual condition there. He had been a decided brunette, with skin darker than usual in his race. There were no areas of pigmentation on the head, face, trunk or extremities. No change was noted in the condition over a period of two years, and presumably the melanoma had been the object of interest thirty years previously (fig. 4).

CASE 2.—A woman, a Russian, aged 32, complained of some asthenopic symptoms. Positive findings consisted of a considerable degree of far-sightedness with astigmatism, in addition to the melanoma of the disk and adjacent choroid of the left eye. There was no enlargement of the blind spot. She was a pronounced brunette with dark skin. There were no areas of pigmentation elsewhere on the head, face, trunk or extremities. In addition to the drawing shown here, Dr. W. B. Doherty [2] had one made at an interval of several years, and a comparison of the two shows no change in the condition (fig. 5).

CASE 3.—A woman, an American, aged 42, sought advice because of some asthenopic symptoms. The refractive error was found to be a moderate amount of hyperopic astigmatism, all other findings being negative except for a melanoma of the disk and adjacent choroid in the right eye. The blind spot showed a negligible extension corresponding to the site of the tumor. The patient was a brunette with a fair skin, and there were no areas of pigmentation elsewhere on her body. Dr. Gordon Bruce, who granted permission to report the case, communicated with the ophthalmologist who had examined the patient three years previously. He had noted the melanoma at that time, and his description of it corresponds to its present appearance (fig. 6).

Sarcoma of the optic nerve has been reported by Vasquez-Barriere,[3] Murray,[4] Cosmettatos,[5] Oloff,[6] Speciale-Cirincione [7] and others, but only three of these cases, those of Vasquez-Barriere, Cosmettatos and Speciale-Cirincione were verified microscopically. These proved to be a primary melanosarcoma of the papilla which arose in the region of the lamina cribrosa. The other cases were reported from the clinical appearance of the lesion with no microscopic substantiation and, therefore, must be viewed with some doubt since the far more common juxtapapillary melanosarcoma of the choroid may appear ophthalmoscopically to arise from the disk. In such cases the growth begins in the choroid adjacent to the disk, proceeds along the course of least resistance around the termination of the lamina vitrea through the intermediary tissue of Kuhnt and lobulates over the internal surface of the

2. Doherty, W. B.: Tr. Am. Ophth. Soc. **26**:309, 1928.

3. Vasquez-Barriere, A.: Ein bemerkenswerter Fall von Melano Sarkom der Papille, Klin. Monatsbl. f. Augenh. **49**:43, 1911.

4. Murray, G. D.: A Case of Sarcoma of the Optic Disc, Ophthalmology **7**:1, 1910.

5. Cosmettatos, G. F.: Sarcome primitif du nerf optique, Rev. gén. d'opht. **37**:5, 1923.

6. Oloff: Ueber primäre Tumoren und tumorähnliche Bildungen der Papilla nervi optica, Klin. Monatsbl. f. Augenh. **55**:313, 1915.

7. Speciale-Cirincione: Sul sarcoma del disco ottico, Ann. di ottal. e clin. ocul. **53**:9, 1925.

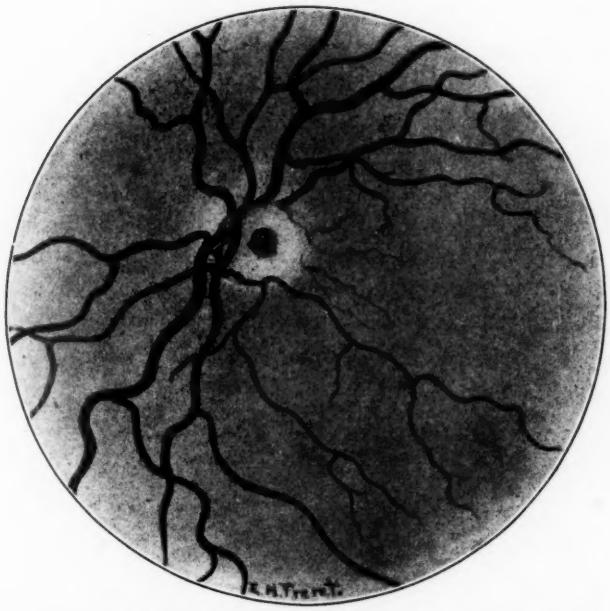

Fig. 4 (case 1).—Melanoma of the disk. The physiologic cup has about the same circumference as the disk. Its entire base is somewhat dark but in the center is a round black pigmented area the nasal border of which is sharp and the temporal border less distinct. It is flush with the surface of the cup.

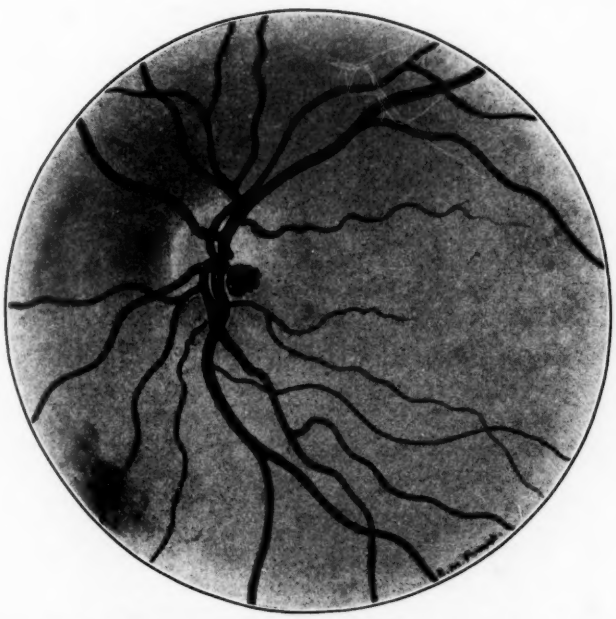

Fig. 5 (case 2).—Melanoma of the disk and adjacent choroid. At the lower outer margin of the disk is a black, very slightly raised (1 diopter), well demarcated pigmented area. The adjacent disk margin and choroid is gray.

optic nerve head. Since the bulk of the tumor and its farthest extension into the vitreous is over the disk, it appears clinically to proceed from the nerve head (fig. 7).

PIGMENTATION OF THE DISK BY PIGMENTED EPITHELIUM

Adjacent to a temporal conus, the pigmented epithelium may extend a short distance into the disk (fig. 8). This may be an actual migration of the pigment into the periphery of the disk, or it may be an extension of the pigmented epithelium into the nerve margin along with the lamina vitrea when the latter sometimes terminates not at the nerve margin but a short distance in it. In such cases the pigment in

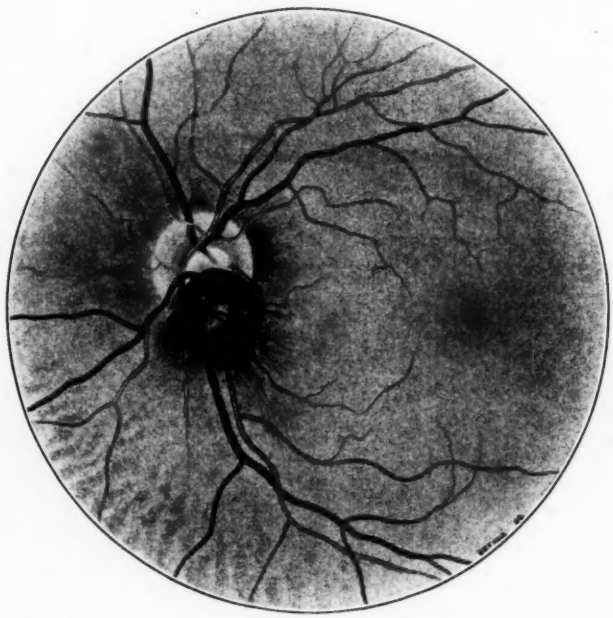

Fig. 6 (case 3).—Melanoma of the disk and adjacent choroid. At the lower temporal margin of the disk is a round, elevated (2 diopter) pigmented mass with a diameter slightly less than that of the disk. It is located half in the disk and half in the choroid.

the disk would be more apparent than real because of the oblique angle at which the nerve inserts into the sclera.

Small, isolated, clearly demarcated dots of jet black pigment are occasionally found located superficially in the nerve fiber layer of the disk (fig. 9). From their color and location they are assumed to originate from the adjacent pigmented epithelium which is often thicker and denser at its termination around the disk, a factor in which may be the tug at the insertion of the optic nerve into the globe occasioned by the movement of the eyeball, since pigmented epithelium proliferates from slight provocation. It is therefore supposed that these spots of

pigment on the disk represent excrescences of pigmented epithelium which have been set free and have migrated into the fiber layer of the disk.

I noted a somewhat similar migration of pigmented epithelium into the disk in a case of Dr. W. B. Doherty's in which a Gonin operation had been performed for detachment of the retina. A reattachment occurred, and in the macular region some pigment was seen. This gradually disap-

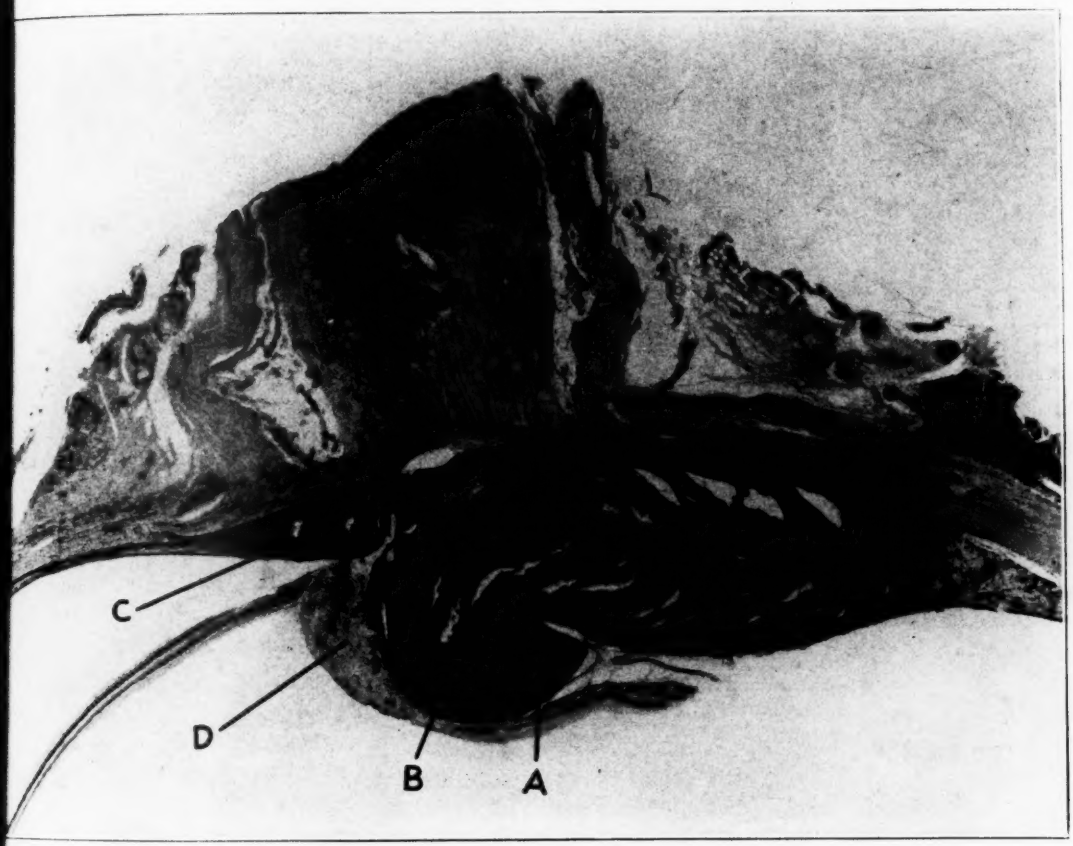

Fig. 7.—A juxtapapillary melanosarcoma of the choroid simulating clinically a growth from the disk: *A*, termination of the lamina vitrea; *B*, the new growth where it has extended around the confines of the lamina vitrea and is growing out into the vitreous anterior to the disk; *C*, extension of the growth in the choroid around the disk to the opposite side; *D*, retina, and *E*, optic nerve.

peared, and in the physiologic cup of the optic disk around the central blood vessels an area of pigment developed.

HEMATOGENOUS PIGMENTATION OF THE OPTIC NERVE

When hemorrhage occurs in the vaginal space around the optic nerve, in the orbit or even in the globe, it may undergo hemolysis with the formation of granules of pigment, which are deposited, either free

Fig. 8.—Pigmented epithelium extending into the periphery of the disk adjacent to a temporal conus.

Fig. 9.—Two areas of pigmented epithelium located superficially on the disk on the temporal side.

or phagocytosed, in the nerve. When this occurs, the pigment is found, not in the stroma of the nerve, but in the interstices of the nerve fiber bundles where vascularity and therefore absorption is at a minimum. This form of acquired pigmentation is in contrast to the previously described congenital pigmentation of the lamina cribrosa in which the pigment is found in the stroma. I have never seen hematogenous pigmentation of the nerve clinically, but a considerable number of cases have been reported in which the condition was observed both ophthalmoscopically and microscopically.

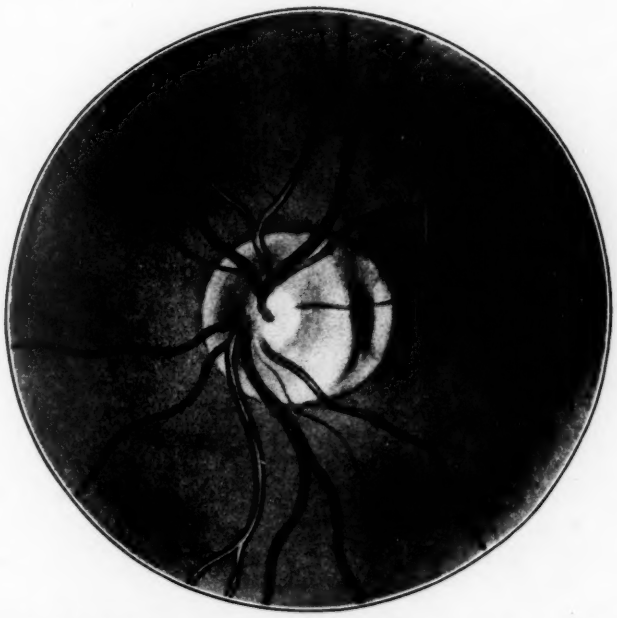

Fig. 10.—An isolated area of pigment at the disk margin in a temporal conus simulating somewhat pigmentation of the disk.

SIDEROSIS OF THE OPTIC NERVE

Although the disk is not one of the sites of predilection for impregnation by iron salts, it may be impregnated in extensive siderosis of the globe. This is probably seldom seen clinically, for by the time siderosis has developed to a sufficient degree to be apparent in the disk the changes in the media, particularly in the lens, are such as to prevent an ophthalmoscopic examination of the interior.

CONDITIONS SIMULATING PIGMENTATION OF THE DISK

1. When the demarcation between the margin of the disk and a temporal conus is indistinct and the pigment of this conus is marked at the periphery of the disk, while the conus area is relatively pigment-

free, it may appear, in a cursory ophthalmoscopic examination, as though the pigment is in the periphery of the disk (fig. 10).

2. A congenital crater-like hole (Grübenbildung) in the nerve head, which is really a form of deep coloboma of the disk not extending through the margin, owes its dark appearance to shadow and to the fact that its base is lined with fibrous tissue. Its appearance is darker with indirect ophthalmoscopy. Its true nature is best observed with direct ophthalmoscopy and with the aid of parallax.

3. The portion of the disk adjacent to medullated nerve fibers often appears dark, especially when the medullation surrounds the disk. This condition which has been reported in the literature as pigmentation of the nerve head is thought to be due to the effect of contrast. The same explanation also perhaps suffices for the grayish appearance of the disk of albinos.

CALCIUM CONTENT AND WEIGHT OF HUMAN CATARACTOUS LENSES

PETER WALDEMAR SALIT, PH.D.
IOWA CITY

Variations in the calcium content of human lenses undergoing cataractous changes require further study for two reasons: (1) the association of cataract with hypoparathyroidism, a pathologic state resulting from insufficiency or absence of the glands which presumably regulate calcium metabolism, discussed extensively by Fischer and Triebenstein,[1] Kerr, Hasford and Shepardson,[2] Eiseman and Luckhardt,[3] Lenhard,[4] Kirby,[5] O'Brien[6] Evans and Kern,[7] and (2) the increase of calcium in cataractous lenses, shown by chemical analyses by Burge,[8] Adams,[9] Updegraff[10] and myself[11] and the joint work of Mackay, Stewart and Robertson.[12] According to these investigators the calcium content of cataractous lenses is extremely variable.

From the Department of Ophthalmology, Dr. C. S. O'Brien, Director, College of Medicine, State University of Iowa.

1. Fischer, J., and Triebenstein, O.: Untersuchungen über Tetanie und Alterstar, Klin. Monatsbl. f. Augenh. **27**:441, 1914.

2. Kerr, W. J.; Hasford, G. N., and Shepardson, H. C.: Treatment of Senile Cataract with Thyroid Extract, Endocrinology **10**:126, 1926.

3. Eiseman, C., and Luckhardt, A. B.: Mechanism of Cataract Formation in Thyroparathyroidectomized Dogs, Proc. Soc. Exper. Biol. & Med. **25**:6, 1927.

4. Lenhard, O.: Linsenregeneration bei Schildrüsenmangel, Arch. f. Ophth. **120**:496, 1928.

5. Kirby, D. B.: A Study of Calcium in Relation to Cataract, Tr. Am. Acad. Ophth., 1930, p. 154.

6. O'Brien, C. S.: The Cataract of Postoperative Tetany, with a Report of Three Cases, Arch. Ophth. **7**:71 (Jan.) 1932.

7. Evans, E. I., and Kern, R.: The Relation of the Parathyroid Gland to Cataract, Am. J. Ophth. **14**:1029, 1931.

8. Burge, W. E.: Analyses of the Ash of the Normal and the Cataractous Lens, Arch. Ophth. **38**:447, 1909.

9. Adams, D. R.: The Rôle of Calcium in Senile Cataract, Biochem. J. **23**: 902, 1929.

10. Updegraff, H.: Calcium, Phosphorus and Cholesterol in Cataractous vs. Apparently Normal Lenses from Human Eyes, Proc. Soc. Exper. Biol. & Med. **29**:964, 1932.

11. Salit, P. W.: Calcium Determinations on Cataractous Human Lenses, Am. J. Ophth. **13**:1072, 1930.

12. Mackay, G.; Stewart, C. P., and Robertson, J. D.: A Note on the Inorganic Constituents of Normal and Cataractous Human Crystalline Lenses, Brit. J. Ophth. **14**:193, 1932.

In a previous publication I [11] pointed out that incipient cataracts contain little or no calcium; however, only slight attention was paid at that time to the exact stage of maturity or to the slit-lamp examinations of the lenses which were analyzed. Furthermore, the results of the previous study were based on lenses extracted by both the extracapsular and intracapsular methods; consequently in some only the nucleus was analyzed, while in others an analysis of the entire lens was made. The present investigation was carried out on whole lenses, i. e., those extracted in the capsule, and with the specific idea of trying to correlate the amount of calcium with the stage of development of the cataract.

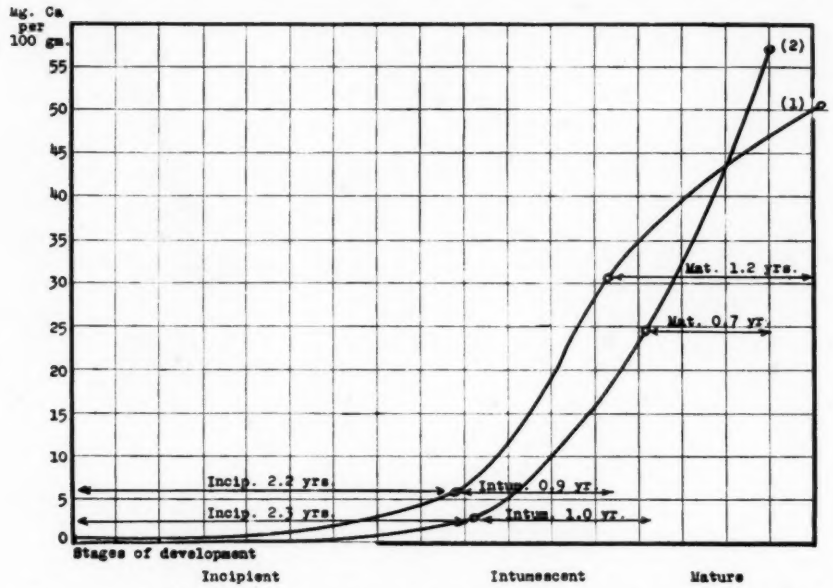

Chart 1.—Calcium values are plotted against the stages of advancement of the cataract. Curves 1 and 2 were obtained on the basis of the classifications according to slit-lamp examinations and the relative calcium contents respectively.

Each lens was examined clinically with the slit-lamp microscope and placed, according to its stage of development, in one of the three following classifications: incipient, intumescent or mature.

On the basis of average values for each of the three groups, the calcium content increases slowly during the incipient stage, then rapidly during the intumescent stage, whereupon the rate of deposition gradually decreases (chart 1, curve 1); the weights of the lenses, on the other hand, decrease uniformly at a slightly accelerated rate (chart 2, curve 1), and the duration of the cataractous condition, according to the history of the case, is directly proportional to the stage of development.

By slit-lamp examination it is not always possible to make an absolute distinction between the three stages, since lesser degrees of swelling are difficult to determine. It is possible that case 28 in table 1, showing a high calcium content, may belong to the class of mature lenses and that cases 20, 31 and 32 in the same table had advanced to the intumescent stage. Likewise, on similar grounds, and supported by the greater than average weights of the lenses, cases 20, 26, 27, 29 and 38 in table 2 may belong to the incipient stage.

TABLE 1.—*Data on Lenses with Incipient Cataracts, Diagnosed by Slit-Lamp Examination*

Number	Age	Wet Weight, Gm.	Calcium, Mg. per Lens	Calcium, Mg. per 100 Gm.	Duration, Years
1	52	0.2272	0.012	5.3	5.5
2	70	0.1813	0.008	4.4	2.0
3	65	0.2145	0.0	0.0	0.5
4	76	0.1700	0.004	2.0	0.5
5	62	0.1564	0.0	0.0	?
6	78	0.2412	0.006	2.5	0.5
7	73	0.2248	0.008	3.5	?
8	76	0.1940	0.012	6.2	0.5
9	45	0.1661	0.0	0.0	1.0
10	62	0.1577	0.004	2.6	?
11	76	0.2017	0.0	0.0	1.0
12	69	0.2233	0.0	0.0	0.5
13	80	0.2236	0.0	0.0	2.5
14	58	0.1653	0.0	0.0	2.5
15	69	0.1849	0.0	0.0	1.0
16	72	0.1977	0.0	0.0	1.0
17	76	0.1427	0.0	0.0	0.5
18	65	0.1770	0.008	4.6	1.0
19	82	0.2633	0.0	0.0	1.0
20	52	0.1891	0.053	28.2	?
21	82	0.1872	0.0	0.0	1.0
22	78	0.2033	0.0	0.0	10.5
23	69	0.2227	0.010	4.5	3.0
24	63	0.2600	0.016	6.1	1.0
25	70	0.2132	0.009	4.4	4.0
26	53	0.2000	0.017	8.7	1.5
27	77	0.2259	0.0	0.0	4.0
28	63	0.1764	0.10	56.7	2.5
29	59	0.1882	0.0	0.0	2.0
30	76	0.2600	0.004	1.5	10.0
31	78	0.2004	0.042	21.0	4.5
32	63	0.2270	0.056	24.7	0.5
33	71	0.1790	0.012	6.7	0.5
34	69	0.2574	0.012	4.7	2.0
35	65	0.1891	0.012	6.30	1.0

For the aforementioned reasons it was considered of some importance to arrange a classification according to the stage of development on the basis of the relative calcium content, for comparison with the chemical classification. In this arrangement, all lenses with a calcium content below 10 mg. per hundred grams were classified as incipient, those with a content within the range of from 10 to 35 mg. as intumescent and those with a content above 35 mg. as mature. According to this classification, in contrast to the first one, each group consists of almost an equal number of lenses, thus: incipient 36, intumescent 35 and mature 32.

The results obtained on the basis of this classification differ considerably from those of the first classification, especially with respect

to the rate of deposition of calcium and the rate of decrease in weight of the lenses with the advancing development of cataract. It appears from chart 1, curve 2, that, once the cataract has made its appearance, calcium tends to accumulate in the lenses quite rapidly at a uniform progressively accelerated rate as the maturation of the cataract advances. The weight of the lenses, on the other hand, decreases gradually up to the intumescent stage, then rapidly during the stage of maturity (chart 2, curve 2). Several investigators have observed a diminution in the weight of cataractous lenses as compared with normal lenses. Clausnizer,[13] on the basis of his own and Grunert's [14] data, found the average weight of thirty immature cataracts to be 0.236 Gm., and that of thirty-six mature cataracts to be 0.213 Gm.; he also reported that the average weight of twelve dislocated transparent lenses was 0.227 Gm., whereas

TABLE 2.—*Data on Lenses with Intumescent Cataract, Diagnosed by Slit-Lamp Examination*

Number	Age	Wet Weight, Gm.	Calcium, Mg. per Lens	Calcium, Mg. per 100 Gm.	Duration, Years
1	34	0.1523	0.040	26.3	3.5
2	59	0.2160	0.048	22.2	6.5
3	45	0.1753	0.028	16.0	2.0
4	74	0.1866	0.040	21.4	2.0
5	76	0.1898	0.036	19.0	1.0
6	63	0.2030	0.034	16.7	3.5
7	48	0.2270	0.030	13.2	1.5
8	54	0.2233	0.080	35.8	3.0
9	35	0.2011	0.034	17.0	5.0
10	88	0.1401	0.096	68.5	7.0
11	78	0.1928	0.020	10.4	1.0
12	39	0.1838	0.040	21.7	1.0
13	77	0.1711	0.064	37.4	1.5
14	68	0.2053	0.037	18.2	4.0
15	71	0.2590	0.065	25.3	1.5
16	54	0.1369	0.057	41.9	3.0
17	61	0.1737	0.105	60.7	2.0
18	64	0.1869	0.112	60.0	2.0
19	72	0.1783	0.109	61.3	2.5
20	73	0.2540	0.012	4.7	4.5
21	71	0.2510	0.037	14.9	0.5
22	75	0.2116	0.038	18.0	3.0
23	78	0.1790	0.068	38.0	0.5
24	76	0.2762	0.046	16.6	2.0
25	75	0.1720	0.046	18.0	14.0
26	79	0.2376	0.0	0.0	1.0
27	61	0.2047	0.0	0.0	3.0
28	62	0.1826	0.146	80.2	5.0
29	70	0.2358	0.013	5.7	2.0
30	26	0.0972	0.049	51.0	5.0
31	72	0.1968	0.049	25.1	7.0
32	48	0.1092	0.025	23.3	2.0
33	73	0.2854	0.072	25.1	0.5
34	83	0.2041	0.068	33.3	4.0
35	51	0.1701	0.052	30.6	3.0
36	44	0.1677	0.060	35.8	4.0
37	71	0.2610	0.057	22.0	1.0
38	76	0.2385	0.016	6.7	2.0
39	72	0.2011	0.068	33.8	0.5
40	72	0.2176	0.101	44.6	5.0

13. Clausnizer, T.: Weitere Ergebnisse von Wägungen der in geschlossener Kapsel entfernten menschlichen Linse, Centralbl. f. prakt. Augenh. **35**:262, 1911.

14. Grunert, K.: Das Gewicht der geschlossener Kapsel extrahierten menschlichen Linse, Centralbl. f. prakt. Augenh. **24**:161, 1900.

that of nineteen dislocated opaque lenses amounted to only 0.183 Gm. Priestly Smith, quoted by Maynard,[15] gave the following average values: immature, 0.239 Gm.; mature, 0.218 Gm. According to Adams[9] the average wet weight of twenty-four normal lenses, from patients aged from 23 to 66, was 0.1348 Gm., and that of thirty-one cataractous lenses, from patients aged from 50 to 77, 0.0987 Gm. Kubik[16] published the following average weights of cataractous lenses: incipient, twenty-nine lenses, 0.206 Gm.; almost mature, one hundred and thirty-eight lenses, 0.209 Gm.; mature, fifty-eight lenses, 0.188 Gm., and hypermature, eighteen lenses, 0.174 Gm. The ages of the patients varied between 38 and 84 years. Mackay and his co-workers[12] stated that the average dry weights of eighteen normal and twenty-seven cataractous lenses were found to be 60.14 and 44.07 mg. respectively; they also submitted the following average values for calcium on the basis of dry weight of the lens: normal, 0.029 per cent; immature, 0.084 per cent, and mature, 0.201 per cent.

On the basis of these findings and other biochemical changes shown by cataractous lenses Mackay and his co-workers concluded that there is a tendency on the part of the lens to return to, or to take on, a composition similar to that of blood plasma, this tendency being shown in immature as well as mature cataracts. If a comparison between normal dog lenses and cataractous human lenses is justifiable, there is a definite increase in the water content of cataractous as compared with normal lenses, the water content, as reported by Evans and Kern,[7] being 61.0 and 64.0 per cent respectively. In fact, these authors found that dog lenses resemble human lenses with respect to their relative weights in normal and cataractous conditions: normal, 0.4690 Gm., and cataractous, 0.4275 Gm. Thus one may say that cataractous lenses undergo a more or less rapid process of liquefaction. A priori, one would conceive of this process as being slow in the beginning, owing to the fact that such liquefaction must be preceded by degeneration or denaturation of the normal lens protein, which in great part is still capable of combating the destructive influences. Once, however, this resistance has been completely broken down, disintegration and resorption of the dead tissue become a one-sided factor; therefore one would expect a cataractous lens to decrease in weight more rapidly in the mature stage. This theory is strikingly supported by the character of chart 2, curve 2.

From the data on duration of the cataractous condition, it appears that, in terms of averages, time is an essential factor in the development

15. Maynard, F. P.: Observations on the Weight, Volume, and Ash of Human Lenses, Brit. J. Ophth. **4**:78, 1920.

16. Kubik, J.: Zur Pathologie menschlicher Stare: Gewichtsverhältnisse menschlicher Linsen, Arch. f. Augenh. **102**:657, 1930.

and maturation of the cataract. This is the more striking when it is considered that estimations concerning the duration of the ailment, as ascertained from the patients, can be, at best, only rough approximations.

In constructing the graphs, the space on the abscissa allotted to each group was determined by the duration of each stage.

In conclusion, it may be said that, despite some points in favor of a classification on the basis of the calcium content as a true index of the stage of development of cataract, this by no means has been conclusively proved in this study. It is hoped that other reports will be published along the same line of investigation from other sources. With

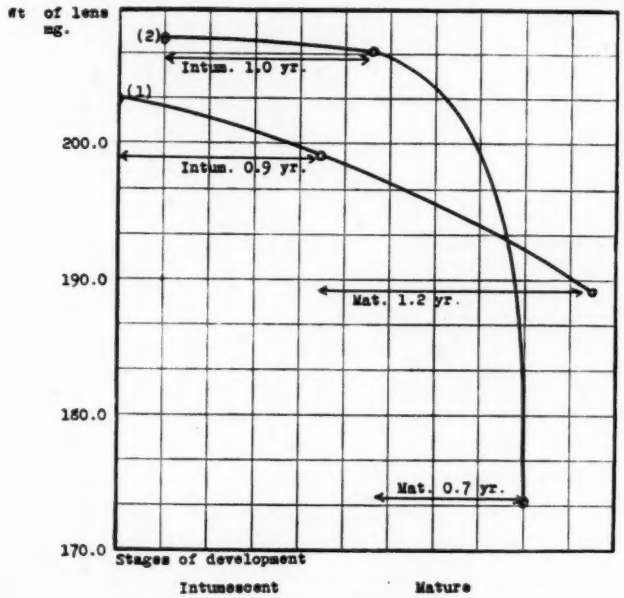

Chart 2.—The wet weights of lenses are plotted against the stages of advancement of the cataracts. Curve 1 is based on the classification as obtained by slit-lamp examinations, and curve 2 is based on the classification according to relative calcium values.

that anticipation in mind the following simple and accurate method for determinations of calcium on small amounts of material, such as a single lens, is outlined:

METHOD

Reagents and Appliances.—These consist of: 1. Approximately tenth-normal potassium permanganate stock solution, standardized against sodium oxalate.[17] This stock solution is kept in the dark. Preceding analysis, 5 cc. of this solution is diluted to 50 cc. in a volumetric flask. The milligrams of calcium, represented by 1 cc. of diluted reagent, are equal to 0.2 × titration factor.

17. Treadwell, F. P., and Hall, W. T.: Analytical Chemistry, New York, John Wiley & Sons, Inc., 1924, vol. 2, p. 98. Hawk, P. B., and Bergeim, O.: Practical Physiological Chemistry, Philadelphia, P. Blakiston's Son & Co., Inc., 1931, p. 460.

2. A saturated solution of ammonium oxalate.

3. One per cent ammonium hydroxide solution (in a wash bottle and kept in the refrigerator).

4. Concentrated ammonium hydroxide (specific gravity, 0.90).

5. An alcoholic solution of 0.05 per cent methyl red.

6. Normal acetic acid.

7. Concentrated perchloric acid (specific gravity, 1.54).

8. Normal sulphuric acid.

9. Fifty cubic centimeter conical centrifuge tubes.

10. A five cubic centimeter Folin microsugar buret.

11. A sand bath with a wire screen (mesh, 1.25 inches [3.1 cm.]) over the top.

TABLE 3.—*Data on Lenses with Mature Cataracts, Diagnosed by Slit-Lamp Examination*

Number	Age	Wet Weight, Gm.	Calcium, Mg. per Lens	Calcium, Mg. per 100 Gm.	Duration, Years
1	71	0.1800	0.068	38.0	3.0
2	80	0.2375	0.088	37.0	5.0
3	69	0.2067	0.066	31.9	0.5
4	69	0.1560	0.082	52.5	2.5
5	63	0.1865	0.080	44.0	3.0
6	72	0.2212	0.072	32.6	?
7	66	0.1750	0.112	64.0	?
8	76	0.1027	0.084	81.8	6.0
9	62	0.1685	0.056	33.2	7.0
10	56	0.1780	0.056	31.5	2.0
11	77	0.1891	0.072	38.0	3.0
12	82	0.2660	0.080	30.0	10.0
13	69	0.1890	0.060	31.7	7.0
14	68	0.2295	0.089	39.0	4.0
15	70	0.2248	0.121	54.0	5.0
16	82	0.2397	0.153	64.0	3.0
17	78	0.1902	0.181	95.4	5.0
18	69	0.2160	0.072	33.3	6.0
19	68	0.2190	0.068	31.0	1.0
20	64	0.1274	0.080	62.8	5.0
21	72	0.2167	0.096	44.2	3.0
22	67	0.1252	0.049	39.4	1.0
23	76	0.1573	0.067	42.8	6.0
24	79	0.2210	0.071	32.3	1.0
25	67	0.1825	0.110	52.3	5.5
26	74	0.0919	0.138	150.6	9.0
27	75	0.1589	0.068	42.8	0.5
28	66	0.1471	0.142	96.8	10.5

Procedure.—The lens is placed on a piece of filter paper (about 20 mm. square) of known weight and weighed immediately on a chemical balance. The lens is then placed in a 50 cc. conical centrifuge tube, the mouth of the tube is covered with filter paper, fastened with a rubber band, and the lens is set aside for analysis. When a convenient number of lenses, e.g., eight or ten, have been collected, 2 cc. of concentrated perchloric acid is added to each tube. The bottoms of the tubes are pressed into the sand of the sand bath to a depth of about 1 inch (2.5 cm.), using the wire screen as a support. The tubes are covered with a large watchglass in order to keep out dust and other foreign materials, and the lenses are digested. The heat of the sand bath is adjusted so that complete digestion of the lenses is accomplished within five or six hours. Digestion is complete when the solutions in the tubes are perfectly clear. After cooling, the sides of each tube are rinsed with about 20 cc. of distilled water from a wash bottle. The contents of each tube are neutralized to methyl red with concentrated ammonium hydroxide. Normal acetic acid is then added until the solution turns pink. This is followed by the addition of 2 cc. of saturated ammonium oxalate solution. After stirring, the

tubes are placed in a refrigerator for three hours and then centrifugated for ten minutes. The supernatant fluid is decanted, and the tubes, still kept in an inverted position, are thoroughly drained on a filter paper. Next, the sides of each tube are rinsed with about 20 cc. of ice-cold 1 per cent ammonium hydroxide solution from the wash bottle and centrifugated. The supernatant fluid is decanted, and the tubes are drained as before. The precipitate in each tube is covered with 10 cc. of normal sulphuric acid, and the tube is placed for five minutes in a 300 cc. beaker containing boiling water. The tube, still remaining in the beaker that has now been removed from the flame, is clamped to a buret holder in such a way that its lower half is surrounded by water and the tip of the buret just reaches into the tube. The potassium permanganate solution (hundredth-normal) is added from the buret, drop by drop, while the contents of the tube are vigorously stirred with a small stirring rod. When the first permanent trace of pink appears, the titration is stopped, the reading is made and 0.2 cc. is subtracted for blank and end-point. Before beginning the titration of the next sample, the beaker containing the tube and solution is heated over a flame for five minutes.

The calculation is as follows:

$$\frac{(\text{cc. permanganate solution} - 0.2\text{ cc.}) \times 0.2 \times \text{factor} \times 100}{\text{weight of lens, Gm.}} = \text{mg. calcium per 100 Gm.}$$

SUMMARY

Calcium and weight estimations were carried out on one hundred and three cataractous lenses, extracted by the intracapsular method, in order to determine the rate of deposition of calcium as well as changes in the weight of the lenses during the formation and maturation of the cataract. If the stages of maturation, as diagnosed clinically by the slit-lamp, are used as a basis for classification, the average calcium values in different stages are: incipient cataract, 5.8 mg.; intumescent cataract, 30.5 mg., and mature cataract, 50.9 mg. per hundred grams. In the same order the average weights of the lenses are 0.2026, 0.1989 and 0.1890 Gm. The average durations of the cataractous changes according to the patients' histories amount to 2.2, 3.1 and 4.3 years respectively. If, however, the cataractous lenses are grouped according to their relative calcium contents in such a way that each group consists of approximately the same number of lenses, the average values are as follows: calcium, 2.5 mg. in incipient, 24.3 mg. in intumescent and 56.6 mg. in mature cataracts; weights of lenses, 0.2075, 0.2066 and 0.1732 Gm. respectively; approximate durations of the cataractous changes, 2.3, 3.3 and 4.0 years respectively. It appears that the relative amounts of calcium may furnish an index of the stage of development.

Dr. C. S. O'Brien, Head of the Department of Ophthalmology, gave valuable help throughout this work. Dr. P. J. Leinfelder assisted in the examination of the patients' histories.

BINOCULAR MOVEMENTS

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NEW YORK

GENERAL CHARACTER AND PURPOSE

Varieties of Binocular Movements.—When one analyzes the movements performed by the two eyes when working together, one is struck by the fact that, with unimportant exceptions, these movements are limited to those subserving binocular fixation.

Thus, in order to produce the binocular fixation of distant objects, the visual lines must be parallel. In harmony with this fact, one finds that there is a whole series of movements—conjugate or parallel movements, or versions—in which the visual line of one eye is kept strictly parallel with that of the other; and, moreover, the vertical meridians of the two corneas also remain parallel, no matter how the eye is directed.[1]

The second great class of associated movements is composed of the disjunctive movements or vergences, in which the visual axis or vertical meridians are made to separate or come together. The vergences are particularly concerned with effecting binocular fixation in near vision.

Associated movements are also classified according to the stimulus that evokes them; e.g., they are divided into voluntary and involuntary

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† Dr. Alexander Duane died on June 10, 1926.

The tables and a large portion of the rest of this chapter are taken from Duane, Alexander: A New Classification of the Motor Anomalies of the Eye Based upon Physiological Principles, Together with Their Symptoms, Diagnosis and Treatment, New York, J. H. Vail.

Mrs. Duane has courteously permitted the publication of certain chapters from the book on "Ocular Muscles," on which Dr. Duane was so industriously working and which he had nearly completed at the time of his unfortunate end. These chapters represent the views of an authority who devoted his life to scientific research in a field to which he had so ably contributed.—THE EDITORS.

1. The exceptions are clearly described by Helmholtz (Handbuch der physiologischen Optik, ed. 2, Leipzig, L. Voss, 1886, p. 631.) He seems to me, however, to have laid too much stress on the ability of the eye to make exceptional movements of this sort, the fact being that such movements are extremely limited and, beyond a certain point, cannot be increased by practice. It seems, therefore, that in spite of his statement to the contrary there is some anatomic basis for the inability of the eye to make unaccustomed movements and that it is not simply a question of training.

(reflex) and also into attraction, avoidance and fusion movements, and they are further classified according to their origin into unicentral and bicentral movements.

CONJUGATE OR PARALLEL MOVEMENTS

Varieties, Versions and Cycloversions.—The conjugate or parallel movements of the eyes comprise duction movements (versions) and parallel torsion movements (cycloversions).

In the parallel duction movements (versions), abduction of one eye is combined with an equal adduction of the other, elevation (superduction) of one eye with elevation of the other and depression (subduction) of one eye with depression of the other.

The two eyes, when performing conjugate movements or versions, move alike; i. e., the movement of one eye equals that of the other, and the two eyes keep pace (comitant movement). The only exception is that in oblique movements of the gaze the in-turning eye, as soon as the object that it is following is cut off from it by the nose, tends to rise somewhat higher in looking up and to sink a little lower in looking down than the out-turning (fixing) eye.

The versions comprise:

Lateriversion, or movements of both eyes to either side, including dextroversion, a movement to the right, and levoversion, or movement to the left.

Elevation (superversion) movements of both eyes up.

Depression (subversion) movements of both eyes down.

Oblique movements combined with lateriversion and elevation or depression.

The versions are performed as the result of an effort, either conscious or unconscious and either reflex or voluntary, set up by a stimulus emanating from the frontal or occipital cortex or from the labyrinth. In this regard it must be noted that lateriversion is set up primarily by a stimulus derived from one side of the brain, but that its complete performance requires the cooperation of both sides. The up and down versions, on the other hand, are produced by a stimulus that is essentially bilateral in origin. The stimuli for the lateriversions are transmitted through the posterior longitudinal bundle; those for the up and down versions, through some other channel, as yet undetermined.

The cycloversions comprise:

Right cycloversion, or a tilting of both vertical meridians to the right (right extorsion combined with left intorsion).

Left cycloversion, or a tilting of both vertical meridians to the left (right intorsion combined with left extorsion).

The cycloversions in all probability do not demand for their production any special nervous mechanism. Normally a cycloversion occurs only when both eyes

are performing an oblique parallel movement and is then the inevitable result of putting into play the muscles required to effect that movement. Thus, as shown in table 1, in looking up and to the right various muscles are called into action, and the necessary result of the contraction of these muscles is to tilt the meridians of both eyes to the right. The right cycloversion in this case is not a specific coordinated movement effected by a special center, but is simply one of the effects of the up-and-right parallel movement. That is, the eyes, receive simply an impulse to move in a straight line, but, owing to the way in which the muscles are inserted, this straight-line movement can not be effected without a tilting of the vertical meridians. Cycloversion, thus, as it normally occurs, is due to peripheral peculiarities and not to central impulses.

TABLE 1.—*Performance of Conjugate Movements*

I. PARALLEL DUCTION MOVEMENTS—VERSIONS

A. *Lateral Movements—Lateriversions*

Both Eyes Move	Muscles Concerned: Right Eye	Muscles Concerned: Left Eye	Move Eyes Laterally*	Move Eyes Vertically*		Rotate Upper End of Vertical Meridians of Cornea		Resultant Effect on Eyes
Right (Dextroversion)	External rectus	Internal rectus	To R	No action		No action		R eye carried to R by the external rectus assisted by the obliques; L eye carried to R by internal rectus assisted by the superior and inferior recti; the other muscles act to steady the eyes and keep them in the horizontal plane; vertical meridians both remain vertical
	Synergists:							
	Superior oblique	Inferior rectus	[To R, action increasing the more eyes are carried to R]	[Down]	Actions equal and opposite	[To L]	Actions equal and opposite	
	Inferior oblique	Superior rectus		[Up]		[To R]		
	Opponents:							
	Superior rectus	Inferior oblique	[[To L] action slight and diminishing as eyes are carried to R]	[Up]	Actions equal and opposite	[To L]	Actions equal and opposite	
	Inferior rectus	Superior oblique		[Down]		[To R]		
Left (Levoversion)	Internal rectus	External rectus	To L	No action		No action		R eye carried to L by the internal rectus assisted by the superior and inferior recti; L eye carried to R by the external rectus assisted by the obliques; the other muscles act to steady the eyes and keep them in the horizontal plane; vertical meridians remain vertical
	Synergists:							
	Superior rectus	Inferior oblique	[To L, action increases the more eyes are carried to L]	[Up]	Actions equal and opposite	[To L]	Actions equal and opposite	
	Inferior rectus	Superior oblique		[Down]		[To R]		
	Opponents:							
	Superior oblique	Inferior rectus	[[To R] action slight and decreases the more eyes are carried to L]	[Down]	Actions equal and opposite	[To L]	Actions equal and opposite	
	Inferior oblique	Superior rectus		[Up]		[To R]		

* Portions enclosed in brackets indicate movements that are completely neutralized.

B. *Movements of Elevation—Superversions* †

Both Eyes Move	Muscles Concerned: Right Eye	Muscles Concerned: Left Eye	Move Eyes Laterally	Move Eyes Vertically	Rotate Upper End of Vertical Meridians of Cornea	Resultant Effects on Eyes
Up	Superior rectus	Inferior oblique	[To L] Actions equal and opposite	Up	[To L] Actions equal and opposite	Both eyes carried vertically up by combined action of superior rectus and inferior oblique; external and internal recti act to steady eye and keep it in vertical plane; vertical meridians remain vertical
	Inferior oblique	Superior rectus	[To R]	Up	[To R]	
	Synergists:					
	External rectus	Internal rectus	[To R] Actions equal and opposite	No action	No action	
	Internal rectus	External rectus	[To L]	No action	No action	
Up and to R	Superior rectus	Inferior oblique	[To L] action slight and decreasing as eyes are carried to R	Up; action marked and increasing as eyes are carried to R	[To L] action slight and decreasing as eyes are carried to R	R eye carried up by superior rectus; L by inferior oblique; R eye carried to R by external rectus assisted (especially in extreme abduction) by inferior oblique; L eye carried to R by internal rectus assisted by superior rectus; both vertical meridians tilted to R
	Inferior oblique	Superior rectus	To R; action moderate and increasing as eyes are carried to R	Up; action slight and decreasing as eyes are carried to R	To R; action marked and increasing as eyes are carried to R; no action	
	Synergists:					
	External rectus	Internal rectus	To R	No action		
Up and to L	Inferior oblique	Superior rectus	[To R] action slight and decreasing as eyes are carried to L	Up; action marked and increasing as eyes are carried to R	[To R] action slight and decreasing as eyes are carried to L	R eye carried up mainly by inferior oblique, L eye by superior rectus; R eye carried to L by internal rectus assisted by superior rectus; L eye carried to L by external rectus assisted by inferior oblique; vertical meridians both rotated to L
	Superior rectus	Inferior oblique	To L; action moderate and increasing as eyes are carried to L	Up; action slight and decreasing as eyes are carried to L	To L; action marked and increasing as eyes are carried to L	
	Synergists:					
	Internal rectus	External rectus	To L	No action	No action	

† In movements of elevation the eyes tend to diverge.

C. *Movements of Depression—Subversion* ‡

Both Eyes Move	Muscles Concerned: Right Eye	Muscles Concerned: Left Eye	Move Eyes Laterally	Move Eyes Vertically	Rotate Upper End of Vertical Meridians of Cornea	Resultant Effect on Eyes
Down	Inferior rectus	Superior oblique	[To L] Action equal and opposite	Down	[To R] Action equal and opposite	Both eyes carried vertically down by combined action of inferior rectus and superior oblique; external and internal recti act to steady eyes and keep them in the vertical plane; vertical meridians remain vertical
	Superior oblique	Inferior rectus	[To R]	Down	[To L]	
	Synergists:					
	External rectus	Internal rectus	[To R] Action equal and opposite	No action	No action	
	Internal rectus	External rectus	[To L]	No action	No action	
Down and to R	Inferior rectus	Superior oblique	[To L] action slight and decreasing as eyes are carried to R	Down; action marked and increasing as eyes are carried to R	[To R] action slight and decreasing as eyes are turned to R	R eye carried down mainly by inferior rectus; L eye by superior oblique; R eye carried to R by external rectus assisted by superior oblique; L by internal rectus assisted by inferior rectus; both vertical meridians rotated to L
	Superior oblique	Inferior rectus	To R; action moderate and increasing as eyes are carried to R	Down; action slight and decreasing as eyes are carried to R	To L; action marked and increasing as eyes are carried to R	
	Synergists:					
	External rectus	Internal rectus	To R	No action	No action	
Down and to L	Superior oblique	Inferior rectus	[To R] action slight and decreasing as eyes are carried to L	Down; action marked and increasing as eyes are carried to L	[To L] action slight and decreasing as eyes are carried to L	R eye carried down mainly by superior oblique; L by inferior rectus; R eye carried to L by internal rectus assisted by inferior rectus; L eye carried to L by external rectus assisted by superior oblique; both vertical meridians rotated to R
	Inferior rectus	Superior oblique	To L; action moderate and increasing as eyes are carried to L	Down; action slight and decreasing as eyes are carried to L	To R; action marked and increasing as eyes are carried to L	
	Synergists:					
	Internal rectus	External rectus	To L	No action	No action	

‡ In movements of depression the eyes tend to converge.

II. PARALLEL TORSION MOVEMENTS—CYCLOVERSIONS

Vertical Meridians of Both Eyes	Muscles Concerned		Acting Specially When Eyes are Directed
	Right Eye	Left Eye	
Straight (vertical)	Superior and inferior obliques	Superior and inferior recti	Straight to R
	Superior and inferior recti	Superior and inferior obliques	Straight to L
	Superior rectus and inferior oblique	Superior rectus and inferior oblique	Straight up
	Inferior rectus and superior oblique	Inferior rectus and superior oblique	Straight down
Tilted to R (R cycloversion; R extorsion + L intorsion)	Inferior oblique	Superior rectus	Up and right
	Inferior rectus	Superior oblique	Down and left
Tilted to L (L cycloversion; R intorsion + L extorsion)	Superior rectus	Inferior oblique	Up and left
	Inferior oblique	Inferior rectus	Down and right

Sherrington's Law as Applied to Conjugate Movements.—Sherrington's law applies to associated movements fully as much as to movements of each eye singly. Hence, when the eyes are to be turned to the right, both right rotators (right externus and left internus) are stimulated, both left rotators (right internus and left externus) are inhibited, and the action of combined abduction of one eye and adduction of the other proceeds according to the plan given subsequently in this article. The modifying factors introduced by the contraction of auxiliary muscles make the movement smoother and more uniform.

The like is true of elevation. If anything—either a voluntary impulse or a reflex action—occurs to make both eyes rise, the four elevators of the two eyes are stimulated, the four depressors are inhibited, and all four lateral rotators are set in action to cooperate in the movement. The reverse action takes place in depression. In both cases the movement proceeds according to the scheme described later.

Muscles Grouped According to Their Main Actions in Performing Conjugate Movements.—Table 1 shows the muscles engaged in performing the conjugate movements. An analysis of the facts presented shows that each of the three main varieties of conjugate movement, viz., lateral rotation, elevation and depression, is effected primarily by a distinct group of four muscles. Each of these groups, which are designated, respectively, as lateral rotators, elevators and depressors, comprises two pairs of muscles, one pair acting predominantly when the eyes are directed to the right, and the other when the eyes are directed to the left. This is shown in table 2.

Associates and Their Fields of Action.—From table 2 it is clear that the twelve muscles of the two eyes, when performing conjugate movements, may be divided into six pairs, one muscle of each pair being in the right eye, and the other in the left eye. The two muscles of each pair are called associates.

With regard to the associates, the following principle holds good: In performing conjugate movements of the eyes each one of a pair of associates has an action similar in all respects to that of its fellow.

Thus, in the case of the right superior rectus and the left inferior oblique, each elevates the eye, and the elevating action increases progressively as the eyes are carried to the right. Each again turns the

TABLE 2.—*Muscles Classified According to Action in Conjugate Movements*

Right rotators	Left rotators
I. *Lateral Rotators* (*Lateriverters*)	
(Carrying both eyes to right)	(Carrying both eyes to left)
R external rectus	R internal rectus
L internal rectus	L external rectus
Right-hand elevators	**Left-hand elevators**
II. *Elevators* (*Superverters*)	
(Acting as elevators mainly when eyes are directed to right)	(Acting as elevators mainly when eyes are directed to left)
R superior rectus	R inferior oblique
L inferior oblique	L superior rectus
Right-hand depressors	**Left-hand depressors**
III. *Depressors* (*Subverters*)	
(Acting as depressors mainly when eyes are directed to right)	(Acting as depressors mainly when eyes are directed to left)
R inferior rectus	R superior oblique
L superior oblique	L inferior rectus

eye to the left and tilts the vertical meridian to the left, and these two actions increase progressively as the eyes are carried to the left. Furthermore, equally in the case of each eye, as these subsidiary actions (left turning, left tilting) increase, the elevating action diminishes, and vice versa. Equally, also, in the case of each eye, all actions performed by these muscles diminish and disappear as the eyes are depressed.

Similar equality of relations obtains in the case of the two muscles constituting each of the other five pairs of associates. Each pair, in fact, acting by itself, would keep the visual lines of the two eyes parallel (and their vertical meridians also parallel) in all directions of the gaze in which the two muscles could act at all.

Table 2 also shows that each pair of associates has its special field of action. That is, there is one special portion of the binocular field of fixation in which the action of each member of the pair is increasingly and equally effective. For the lateral (right and left) rotators,

this field is the right or the left half of the total binocular field. For the elevators and depressors, it is the upper or lower half of the complete field, but so far as the main actions are concerned, it is limited to a single quadrant, and so far as the subsidiary actions are concerned, it is limited to another quadrant which is contralateral to that in which the main action is most effective. These facts are indicated in figure 1.

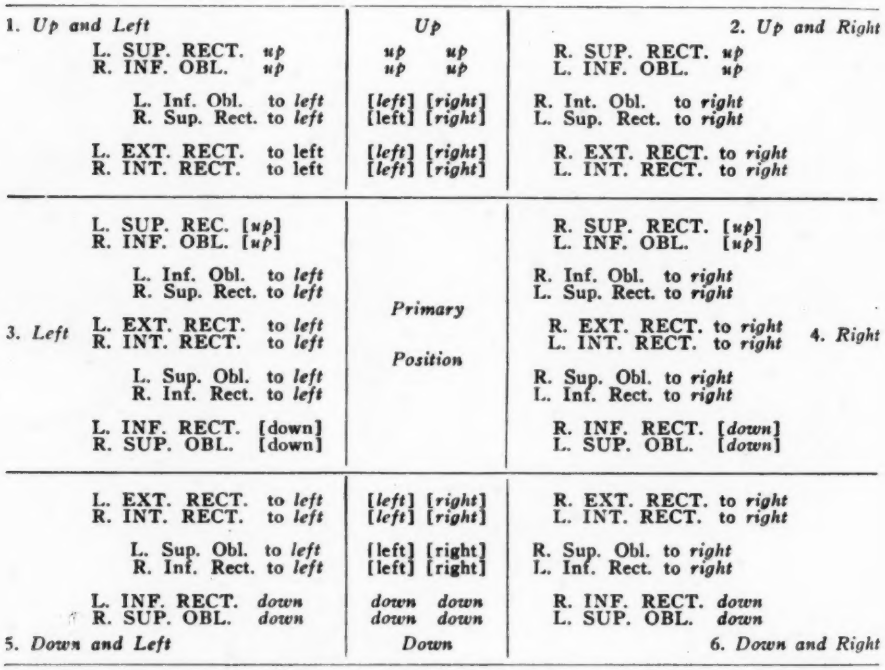

Fig. 1.—For the nomenclature used in this diagram and for its explanation see figure 1 in a previous article,[2] from which, in fact, it is derived by substituting right or left for out and in, as the case requires. The six fields of action (six diagnostic directions of the gaze) are numbered *1, 2, 3, 4, 5* and *6*. The diagram shows that when the eyes are directed straight to the right (field of action *4*), the muscles effecting the movement are the right external rectus, assisted by the right superior and inferior obliques, and the left internal rectus, assisted by the left superior and inferior recti. All four elevators and depressors cooperate in the movement, but the vertical actions of these muscles are either absent or neutralized.

It must be pointed out with regard to figure 1 that, as in the case of figure 1 in a previous article,[2] it does not represent the facts with rigorous precision. That is, the fields of action do not stop at the midline, but pass it somewhat, so that the field of the left-hand elevators (field *1*) overlaps that of the right-hand elevators (field *2*), the left-hand rotators (field *3*) and the left-hand depressors (field *5*).

2. Duane, Alexander: The Monocular Movements, Arch. Ophth. **8**:530 (Oct.) 1932.

The diagram shows clearly the similarity in action of the two muscles forming each pair of associates. The same thing may be shown by figure 2 of the previous article.[1] Thus, if alongside of figure 2, which represents the field of fixation of the right eye, one places one representing the field of fixation of the left eye (which may be done by turning the figure end for end, so that *O* is on the left and *P* on the right of the figure), one will see how the field of action ($A_2B_2C_2D_2E_2$) of the inferior oblique of the right eye agrees in all respects with the field of action of the superior rectus of the left eye. So also of the other associates (right superior rectus and left inferior oblique; right inferior rectus and left superior oblique; right superior oblique and left inferior rectus).

The Six Diagnostic Directions of the Gaze in Binocular Vision.—Just as in monocular vision, so also in binocular vision, there are six diagnostic directions of the gaze, each corresponding to a single pair of associates. This is shown in figure 1, in which the diagnostic directions are indicated by numbers from *1* to *6*. The directions, with the muscles belonging to each area, are as follows:

Diagnostic Directions (Fields of Action)	Muscles Chiefly Acting Therein (Comprising the Six Pairs of Associates)	
	Right Eye	Left Eye
Right	External rectus	Internal rectus
Left	Internal rectus	External rectus
Up and right	Superior rectus	Inferior oblique
Up and left	Inferior oblique	Superior rectus
Down and right	Inferior rectus	Superior oblique
Down and left	Superior oblique	Inferior rectus

This tabulation expresses in brief the cardinal principle in the diagnosis of paralyses of the ocular muscles.

Limits of Binocular Excursions; Clinical Importance of Their Determination.—Much more than in the case of monocular vision is it important to determine the range of excursion when the two eyes act together, for it is a matter of comparatively little importance to a man how far he can move one eye in a given direction, provided the other eye moves just as readily and just as far. Ophthalmologists see this fact often exemplified in practice. Many persons have more or less restriction in the ability to move the eyes upward. Yet they seem to suffer little inconvenience, provided both eyes keep pace with each other and both fail simultaneously. On the other hand, it not infrequently happens that a patient has full and equal movement in a given direction with each eye when tested separately, yet when the two eyes are working together, one lags behind the other and thus gives rise to a corresponding and disagreeable diplopia. This finding which I have met with several times, must mean that while one eye, if tested by itself, can make a maximum effort equal to that of the other, when it works with its fellow it fails to make an equal effort and does so because one of its muscles is at least relatively weak. Such a relative weakness may cause uncomfortable symptoms.

In the estimation of the range of binocular excursions one is, in fact, concerned not so much with their maximum extent, although this is important, as with the way in which the two eyes keep pace with each other in performing them. This question is determined by ascertaining if and when in making the excursions one eye lags behind the other and if and when diplopia develops.

Thus, measurements of the excursions of the eyes forming conjugate movements consist in the determination of three facts: the field of binocular fixation; the field of binocular excursion, and the field of binocular single vision.

Field of Binocular Fixation and Field of Binocular Excursion.—The field of binocular fixation is delimited by ascertaining for each direction of the gaze the point at which either one of the eyes fails to follow an object moving before the two. This can be done conveniently with the double dot used for testing the monocular field of fixation, since the moment when either eye fails to follow the dots or when either eye fails to keep up with the other in following them is rendered evident by a blurring of the image causing the two dots to run into one.

Hering, who used a different method (with after-images), found the binocular field to be of quite small extent, being considerably smaller than the portion common to the two monocular fields.

It seems likely, however, that his tests in this case, as in the case of the monocular field, were made on a not altogether normal subject. My own researches,[3] although few, to be sure, were made on quite normal persons. They gave the following results:

Movements of Both Eyes

	Up	Up and Right	Up and Left	Right	Left	Down	Down and Right	Down and Left
Case 1.....	38	52	45	58	52	70	80	70
Case 2.....	50	52	53	56	59	70	62	70

The field of fixation here described is the transverse field, but there is also a longitudinal field of binocular fixation.

The field of binocular excursion is larger than the field of binocular fixation in every direction except straight down, since in other directions the gaze, though the eyes move perfectly together, fixation is cut off by the nose or the brow.

Field of Binocular Single Vision.—The field of binocular single vision is determined by noting in any particular direction of the gaze the point at which one eye can no longer keep pace with the other, as evidenced by the development of an insuperable diplopia.

3. Examination was made for near points.

The field defined by joining all such points is not necessarily coincident with the field of binocular fixation, since it is quite conceivable that the two eyes following a moving object might fail to fix it, but might yet both lag behind to an equal extent, so that the two images, although not formed on the maculae, would still be formed on corresponding points. In this case, binocular single vision would still be present, although binocular fixation would no longer exist.

A point on which some stress has been laid is that this method of delimiting the field of fixation gives uncertain results, since, as is alleged, many people fail to recognize diplopia in eccentric positions of the gaze. It is claimed, in other words, that the normal field of binocular single vision is quite small, and that diplopia occurs normally in looking far up, far to the right, etc., but that its existence is not suspected, because the subject under examination either fails to notice or actually suppresses one image. My own experiments, however, lead me to negative this idea completely. If one employs a candle for the test object and places a red glass before one eye of the person examined, the presence of binocular single vision will be shown by the fact that the candle flame appears pinkish or, more commonly, yellow with a reddish border. Manifest diplopia will be shown by the presence of two flames, one red and the other yellow, and diplopia with the suppression of either image by the presence of one flame, either pure red or pure yellow. The differences presented are marked and readily appreciated by an observing patient when once they have been pointed out to him.

Testing in this way a large number of people with apparently normal eyes, I have uniformly found that the field of binocular single vision extends not less than 40° in any given direction and usually extends up to 50° or more. Indeed, most persons still get true binocular single vision, even when the eyes are carried to the extreme limit of their excursion, the field of binocular single vision being larger than either monocular field of fixation taken separately. This is but another instance of the law that the movements of the eyes, however extensive or however limited in themselves, are always under normal conditions modified in such a way as to best subserve binocular fixation and binocular single vision. Thus, as the experiments just adduced seem to show, it appears that, no matter what the maximum range of excursion of each eye separately is, the excursion of both together, effected by the coordinating action of the association centers, is in normal cases such that one eye keeps pace with the other, going neither faster nor slower, and that each stops moving when the other does. Hence, however far the object looked at may be carried in any given direction, no diplopia occurs, or, if it does, it is transient and superable.[4]

4. These statements presuppose (1) that the visual lines are not far from parallel, i. e., the test object should not be less than 30 inches (76.2 cm.) from the eyes, and (2) the person examined should endeavor all the time to follow the object, i. e., must not look beyond it. In the latter case, of course, the test object will seem to him double. Such diplopia is, however, usually at once superable by voluntary effort.

A parallel movement is frequently associated with a movement of convergence. (On this point see the section on "Vergences".) Here it need only be mentioned that whenever the eyes are elevated they tend to diverge and whenever they are depressed they tend to converge. To this fact, which has some clinical significance, I shall refer repeatedly later.

VERGENCES [5]

Varieties of Vergences.—The vergences are movements that cause the visual lines or the vertical meridians of the two eyes to turn toward each other or when so turned to separate again. The impulse that effects their mutual approach or separation seems to be always equal for the two eyes.

The vergences include:

Convergence.
Divergence.
Supervergence (divergence or convergence in a vertical plane).
Convergent or divergent torsions.

Convergence.—Convergence consists in a simultaneous contraction of the two internal recti, thus causing both eyes to turn in toward an object nearer than the one already sighted. So far as can be told, the impulse to this action is always precisely equal for the two eyes. It may often seem unequal, but when it does so, it will be found that the convergence is associated with a conjugate movement which makes one in-turning eye turn out again and the other turn still farther in. Thus, in looking at a near object situated to the left, there is a conjugate movement which carries both eyes equally far to the left and with this a contraction of both internal recti which neutralizes in part the left-hand movement of the left eye and reinforces the left-hand movement of the right eye. The same thing occurs when both eyes are fixing an object and a prism, base out, is placed before one of them, say the right. The eye behind the prism turns sharply in; the other apparently remains straight. In reality, however, both eyes move equally. For example, suppose that the two eyes are fixing an object, *A*, and a prism with a deviating power of 10° is placed, base out, before the right eye. This prism deflects the retinal image of *A* in the right eye 10° to the right. To get this image on the fovea and so

5. In recent editions of Fuch's "Textbook of Ophthalmology" I have given the name of "disjunctive movements" to the vergences. However, the term "disjunctive" is not the antithesis of "parallel" or "conjugate" but of "associated," and is therefore more properly applied to the dissociated movements that oppose fusion than to convergence and divergence, which subserve it.

reacquire single vision, the eye must turn 10° to the left. But if it turned to the left by a simple conjugate movement, the left eye would necessarily turn too and by an equal amount, so that it would look 10° to the left of *A*. On the other hand, if the right eye turned to the left by a simple movement of convergence, the left eye would necessarily turn to the right by an equal amount, i. e., it would point 10° to the right of *A*. As a matter of fact, however, both eyes move 5° to the left by a conjugate impulse and then both converge 5°. In so doing each receives the image of *A* on its fovea, so that there is binocular single vision. In the case of the left eye, the right-hand movement due to convergence just offsets the left-hand movement due to conjugate action, so that the eye apparently does not turn at all, but remains in the fixing position. The right eye, on the other hand, turns 5° to the left by conjugate action and then 5° more by convergence, so as to be pointed 10° to the left of its original position and thus fixate the displaced image of *A*.

The effect of this convergence effort, shared equally by both eyes, is shown by the fact that each accommodates more or less strongly when a prism, base out, is placed before one of them. A mere side-to-side movement would not set up such an accommodative action.

Longitudinal, Transverse and Total Fields of Binocular Fixation.—Since the eyes can converge through a wide range as well as travel extensively from side to side and up and down, they can perform binocular fixation on all points contained in a segment of space which extends widely from side to side and, fore and aft, from far distance down to the convergence near point. This segment of space constitutes the total field of binocular fixation. The extent through which the two eyes, when both are fixing the same object, can range from side to side and up and down delimits what has already been called the transverse field of binocular fixation. Similarly, the extent through which they can range fore and aft, when continuing to converge and fix, delimits the longitudinal field of binocular fixation. It may be said, then, that the transverse field of fixation is formed by the intersection of the total field of binocular fixation by a plane transverse to the bivisual line, and that the longitudinal field of fixation is formed by the intersection of the total field by a fore-and-aft plane, i. e., a plane coinciding with the bivisual line. It is apparent that there will really be any number of transverse fields of binocular fixation, depending on the distance of the object fixed. There will also be any number of longitudinal fields, depending on the direction of the bivisual line.

Nature of Convergence; Difference Between Convergence Adduction and Conjugate Adduction.—The inward movements that each eye

makes when converging with its fellow are called convergence adduction. Convergence adduction is to be distinguished from conjugate adduction (latero-adduction), which consists of a movement inward of one eye associated with an equal outward movement of the other, so that the visual lines remain parallel. Convergence adduction and conjugate adduction are effected by the same muscles, i. e., the internal rectus, associated probably in both cases with the superior and inferior recti. The nervous mechanisms governing the two are quite distinct. Consequently, one function may be out of gear without any disturbance of the other. Thus, there may be a paralysis in which the eye cannot turn in in performing a side to side movement and yet may turn in well when converging with its fellow (paralysis of conjugate adduction), and there may also be just the reverse (paralysis of convergence with conjugate adduction intact). Furthermore, convergence adduction is regularly associated with a corresponding effort of accommodation and with a contraction of the pupil (convergence reaction), while with simple conjugate adduction no such association takes place.

The pupillary contraction and accommodative effort associated with convergence occur when a convergence effort is made, even if that effort is nullified by an outward movement due to conjugate action so that the eye does not turn in, but stands still.

Convergence regularly occurs as the result of an effort, conscious or unconscious, put forth in order to bring both eyes in on a visible object to which the attention is directed. Ordinarily it cannot be initiated by a mere effort of the will apart from any attracting object. A conjugate movement can be so initiated. Thus, almost any one, by an effort of the will, can move the eyes up, down or laterally without having any object to look at. But there are few persons indeed who can voluntarily turn both eyes inward simultaneously unless stimulated to do so by the attraction of a visible object. Convergence is thus usually an attraction movement and the result of stimulation of the occipital cortex; rarely, it is the result of an impulse emanating from the frontal centers.

To the rare persons who can converge in at will, objects more distant than the point at which both eyes are directed appear double (homonymous physiologic diplopia of distant objects), and in their case the diplopia is more apparent than usual, because there is no real object of fixation to distract their attention. It is in this way that some persons have the ability to produce homonymous diplopia at will.

The ability to converge without an object to look at and thus to produce homonymous diplopia voluntarily is not necessarily indicative of a high converging power. Rather it indicates a high imaginative power which makes the eyes attempt to fixate something nonexistent but in some way conceived of as possibly existing.

Convergence, when marked, modifies somewhat the effect of the other muscles that are acting with the interni. Thus, when the gaze is directed at a near object in the median line, the superior rectus of one eye and the inferior oblique of the other no longer act as associates, the former now serving mainly to adduct, and the latter to elevate the eye. In this case, in fact, the superior and inferior obliques in each eye neutralize each other completely, and the two superior and two inferior recti act as synergists to the two interni, all adducting the eye. Again, when the gaze is directed at a very near object, situated upward and to the right, the right superior rectus, since the right eye is not pointed as far to the right as the left one is, will not be working as an elevator at quite the same mechanical advantage as does the left inferior oblique. Thus, the right eye will lag somewhat below the left. In general, when the eyes are directed sideways and up or down at a near object, the in-turning eye will rise higher in elevation and drop more in depression than the out-turning fellow.

Convergence, when of considerable amount, is associated with a divergence of the vertical meridians of the two eyes.

Measurement of Convergence.—Convergence may be measured in three principal ways. To understand this better, several terms should be defined.

The convergence angle is the angle through which each eye turns in performing convergence adduction. It is the true measure of the convergence action.

The convergence distance is the distance from the eye of the point toward which the two eyes are converging. The distance should be measured from the base line, connecting the centers of rotation of the two eyes.

The convergence near point (*Pc*) is the point toward which the eyes are directed when converging as much as possible. The convergence distance in that case, now at its maximum, may be called the *PcB,* because the *Pc* is measured from the base line.

The convergence distance may be measured in millimeters or in meter-angles. The meter-angle (*MA*) is the amount of convergence put forth when both eyes are converging to 1 meter. Two meter-angles denote a convergence adduction for half a meter; one-half a meter-angle, the convergence adduction for 2 meters, etc.

The prism convergence is expressed by the deviating power of the prisms, base out, which the patient can overcome by turning the eye in.

Relations Between Convergence Distance and Convergence Angle.—The convergence distance, whether measured in millimeters or in meter-angles, is an inexact measure of the convergence. Persons having the same convergence distance will have a different convergence angle, according as their interaxial distances differ. Obviously, a man with an interaxial distance of 65 mm. will have to converge a good deal more when looking at an object 60 mm. away than will a child

with an interaxial distance of 55 mm. This is clear from figure 2, in which *TPC* is the convergence angle that may be denoted by *A*. It is evident that the greater *TC* is, *PC* remaining the same, the greater will be the angle *TPC* or the convergence. *TC* is practically the same as *CO*, which is half the interaxillary distance.

That *TCP* really represents the convergence angle is shown in the following description:

Draw *AI* perpendicular to *M'N'*, and prolong *AO* so as to intersect *M'N'* in *T*. Then in the triangles *MNA* and *M'N'A* the sides are respectively equal (*MN* = *M'N'*, *AM* = *AM'*, *AN* = *AN'*). Hence, the triangles are equal, and the angle *ANM* equals the angle *AN'M'*.

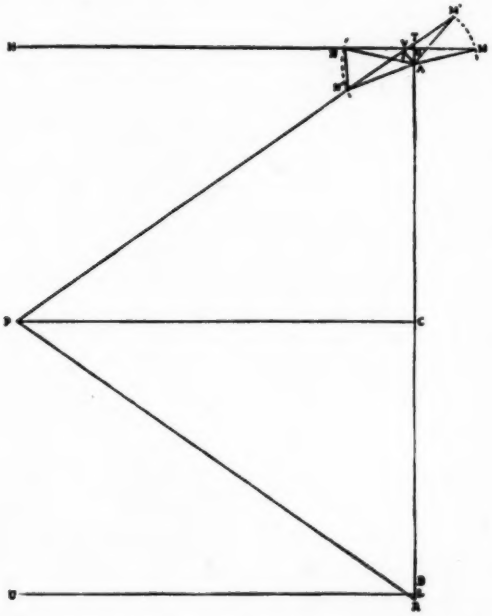

Fig. 2.—Relation between the convergence near point and the convergence angle. *A* indicates the center of motion of the right eye; *B*, the center of motion of the left eye. *C* indicates the midpoint of the intercentral line; *P*, the point on which the eyes converge. *M* indicates the macula and *N*, the nodal point of the right eye when the latter is looking straight ahead; *MONH* is the visual line when the eye is thus directed. *M'* indicates the macula, *N'*, the nodal point and *M'TN'P*, the visual line when the eye is converged to *P*. *U* indicates the visual line of the left eye when looking straight ahead; *RP*, the visual line when converging on *P*. *NAN'* equals *MAM'*, the angle of rotation (convergence adduction). As shown in the text, this equals angle *TPC*. The converging lines of the two eyes (*TP* and *RP*) do not start, as is usually assumed, from the centers of motion of the two eyes (*A* and *B*), but from a point from 1.7 to 2 mm. or more further out (*T* and *R*). Practically, however, for all ordinary cases they may be regarded as starting from *O* and *L*, respectively. It is thus seen that the interaxial distance, *OL*, and not the intercentral distance, *AB*, is the important thing to measure in determining the convergence angle.

Assuming, as is true for all ordinary rotations, that the center of rotation remains fixed, and that the relations are as indicated by George [6] and others, figure 2 illustrates what happens.[7]

Let *M* be the macula and *N* the nodal point of the right eye when in the primary position. *MNH* then represents the visual axis when the eye is looking straight ahead. *A*, the center of rotation of the eye, lies at a distance, *AO* equals 1.65 mm., to the nasal side of *MN*. *B* is the center of rotation of the left eye and *LU* the visual axis of the left eye, when looking straight ahead. *ACB* is the base line connecting the centers of rotation of the two eyes, *C* being its midpoint. *CP* is the median line of the head.

TABLE 3.—*Relation Between the Convergence Angle and the Convergence Distance*

Convergence Angle	Convergence Distance for Intercentral Distances of											
	55	57	58	59	60	61	62	63	64	65	67	69
40	33	34	35	35	36	36	37	38	38	39	40	41
39	34	35	36	36	37	38	38	39	40	40	41	43
38	35	36	37	37	38	39	40	40	41	42	43	44
37	36	37	38	39	40	40	41	41	42	43	44	46
36	38	39	40	40	41	42	43	43	44	45	46	47
35	39	40	41	42	43	44	44	45	46	46	48	49
34	41	42	43	44	44	45	46	47	47	48	50	51
33	42	44	45	45	46	47	48	49	50	50	52	53
32	44	46	46	47	48	49	50	50	51	52	54	55
31	46	47	48	49	50	51	52	52	53	54	56	57
30	48	49	50	51	52	53	54	54	55	56	58	60
29	50	51	52	53	54	55	56	57	58	59	60	62
28	52	54	55	56	56	57	58	59	60	61	63	65
27	54	56	57	58	59	60	61	62	63	64	66	68
26	56	58	59	60	62	63	64	65	66	67	69	71
25	59	61	62	63	64	65	67	68	69	70	72	74
24	62	64	65	66	67	69	70	71	72	73	75	77
23	65	67	69	70	71	72	73	74	75	76	79	81
22	68	70	72	73	74	75	77	78	79	80	83	86
21	72	75	76	77	78	80	81	82	83	85	87	90
20	76	79	80	81	83	84	85	87	88	89	92	95
19	80	83	84	86	87	89	90	91	93	94	97	100
18	85	88	89	91	92	94	95	97	98	100	103	106
17	90	93	95	96	98	100	101	103	105	106	109	113
16	96	99	101	103	105	107	108	110	112	113	117	120
15	103	106	108	110	112	114	116	118	119	121	125	129
14	110	114	116	118	120	122	124	126	128	130	134	138
13	119	123	126	128	129	132	135	136	139	141	145	149
12	129	134	136	139	141	143	146	148	150	153	158	162
11	141	146	149	152	154	157	160	162	165	167	172	178
10	156	162	164	167	170	173	176	179	182	184	190	196
9	174	180	183	186	189	193	196	199	202	205	211	218
8	199	203	206	210	213	217	221	224	228	232	239	246
7	224	232	236	240	244	248	253	257	261	265	273	281
6	262	271	276	281	285	290	294	300	305	309	319	329
5	314	326	331	337	344	349	354	360	366	371	383	394
4	393	408	415	422	429	436	444	451	458	465	479	494
3	525	544	554	563	571	582	591	600	610	619	628	659

When, now, the right eye is rotated in so as to be directed at a point *P* of *CP*, the nodal point *N* swings inward to *N'*, and the macula *M* outward to *M'*, about *A* as a center, and the visual axis assumes the position *M'N'*, intersecting its original position *MN* in the point *V*. *M'*, *N'* and *P* are in a straight line. The angle of rotation, which represents the convergence angle of the eye, is *NAN'*.

6. George, E. J.; Toren, J. A., and Lowell, J. W.: Am. J. Ophth. **6**:833 (Oct.) 1923.

7. It makes no difference in the essential accuracy of what follows whether these relations are precisely as indicated or not. Whatever they are, the relation between the position of the convergence near point and the amount of convergence adduction remains the same.

Again, in the triangle VNN' the angle $VNN' = ANN' + ANM$, and the angle $VN'N = AN'N - AN'M' = AN'N - ANM$. Hence $VNN' + VN'N = ANN' + AN'N$. Accordingly, angle $NAN' = 180° - (ANN' + AN'N) = 180° - (VNN' + VN'N) =$ angle $NVN' = TPC$.

In other words, the angle through which the eye rotates about the eccentric point A in order to converge on P is equal to the angle TPC, which thus represents the true convergence angle of the eye, which I have denoted by a. The tangent of this angle is represented by $\frac{TC}{PC}$. T is so close to O (even in extreme degree of convergence being less than 0.5 mm. from it) that without sensible error it may be said that $a = \frac{CO}{PC}$. But CO is one-half the distance between the usual axes of the two eyes (interaxial distance), and PC is the distance of the convergence near point P from the base line AB, connecting the centers of rotation of the two eyes (convergence distance).

For all values of a practically met with, the following simple relation holds good:

$$a = \frac{2CO \times 25}{PC} + 1.5°$$

or the angle by which an eye converges on an object is found by multiplying the interaxial distance by 25, dividing it by the convergence distance, and adding 1.5°. If, for example, the interaxial distance is 60 mm., and the convergence distance is 75 mm., the angle through which the eye is converging is

$$\frac{60 \times 25}{75} + 1.5° = 21.5°$$

This will hold for all values of a from 11 to 30° or, with eyes of average interaxial distance, for all convergence distances from 130 to 55 mm. For distances less than this (from 55 to 50 mm.) [8] 1° should be added to the fraction instead of 1.5°. If the distance is greater than 130, the following additions should be made:

Distance of object 130-300 mm	add 1°
Distance of object 400-600 mm	add 0.5°
More remote points	add 0°

Thus, if the eyes having an interaxial distance of 55, are converged on an object at 150 mm., the adduction of each would be

$$\frac{55 \times 25}{150} + 1° = 10°$$

If directed at an object 2 meters distant, they would each be converging

$$\frac{55 \times 25}{2000} + 0° = 0.7°$$

One fact deduced from the foregoing demonstration deserves to be especially emphasized, because it runs counter to the statements and particularly to the illustrations contained in some textbooks. When the two eyes converge on a point such as P, so that their visual axes meet in the latter, these axes do not, as is often represented, pass through the centers of the rotation of the eyes (A and B). On the contrary, they intersect the intercentral base line in points (T and R) 1.6 mm. and more to the temporal side of the centers. Moreover, the angle by which the

8. But when a exceeds 30° all calculations become rather uncertain on account of the probability of error in the measurement and also because of the probable shifting that now takes place in the center of rotation.

eyes converge is not, as is also usually represented, that between the lines drawn from P to the two centers of rotation A and B, but between the lines drawn from P to the two points T and R, or practically to the two points O and L. That is, it is the interaxial and not the intercentral distance that one has to consider in making measurements and in determining the convergence angle.

In order to avoid calculation and make the determination of the convergence adductions from the convergence distance directly in all cases, I have computed table 4.

Relation Between Meter-Angle and the Convergence Angle.—The supposed advantage of the meter-angle is that it indicates the relation between convergence and accommodation. Thus, a man who is exert-

TABLE 4.—*Values of Meter-Angles*

Meter-Angles	Corresponding Convergence Angle for Interaxial Distance of								Convergence Distance in Mm.
	56	58	60	62	64	66	68	70	
1	1.6°	1.7	1.7	1.8	1.8	1.9	1.9	2.0	1,000.0
2	3.2	3.3	3.4	3.5	3.7	3.8	3.9	4.0	500.0
3	4.8	5.0	5.1	5.3	5.5	5.7	5.9	6.0	333.3
4	6.4	6.6	6.9	7.1	7.3	7.5	7.7	8.0	250.0
5	8.0	8.2	8.5	8.9	9.2	9.4	9.6	9.9	200.0
6	9.5	9.9	10.2	10.5	10.9	11.2	11.5	11.9	166.7
7	11.1	11.5	11.9	12.2	12.6	13.0	13.4	13.8	142.8
8	12.6	13.1	13.5	13.9	14.4	14.8	15.2	15.6	125.0
9	14.1	14.6	15.1	15.6	16.1	16.5	17.0	17.5	111.1
10	15.9	16.2	16.7	17.2	17.7	18.3	18.8	19.3	100.0
11	17.1	17.7	18.3	18.8	19.4	19.9	20.5	21.1	90.9
12	18.6	19.2	19.8	20.4	21.0	21.6	22.2	22.8	83.3
13	20.0	20.7	21.3	21.9	22.6	23.2	23.8	24.5	76.9
14	21.4	22.1	22.8	23.5	24.1	24.8	25.5	26.1	71.4
15	22.8	23.5	24.2	24.9	25.6	26.3	27.0	27.7	66.7
16	24.1	24.9	25.6	26.4	27.1	27.9	28.5	29.2	62.5
17	25.5	26.2	27.0	27.8	28.5	29.3	30.0	30.8	58.8
18	26.7	27.6	28.4	29.2	29.9	30.7	31.5	32.2	55.6
19	28.6	28.9	29.7	30.5	31.3	32.1	32.9	33.6	53.6
20	29.2	30.1	31.0	31.8	32.6	33.4	34.2	35.0	50.0
21	30.5	31.2	32.2	33.1	33.9	34.7	35.5	36.2	47.6
22	31.6	32.5	33.4	34.3	35.1	36.0	36.8	37.6	45.5
23	32.8	33.7	34.6	35.5	36.4	37.2	38.0	38.8	43.5
24	33.7	34.8	35.8	36.6	37.5	37.5	38.4	40.0	41.7
25	35.0	36.0	36.9	37.8	38.7	39.5	40.4	41.2	40.0

ing a convergence of 3 meter-angles—representing a convergence distance of 333 mm.—is supposedly exerting at the same time an accommodation of 3 diopters.

As a matter of fact, the converging power so greatly exceeds the accommodating power that there is no proper relation between meter-angles and the number of diopters of accommodation that the patient is able to exert. Moreover, the meter-angle, like the convergence distance of which it is the reciprocal, gives but an approximate idea of the work done in converging, especially at very near points. The amount of this work is represented by the convergence angle, and two persons converging on a point equally remote and converging, therefore, through the same number of meter-angles will differ in the convergence angle and consequently in converging power, unless their interaxial distances

are identical. Thus, 12 meter-angles may represent a convergence angle of anywhere from 19 to 22°, and 20 meter-angles a convergence angle of from 30 to 35°.

Usual Values of the Convergence Distance and of the Convergence Expressed in Meter-Angles.—The convergence distance (*PcB*), i. e., the distance of the converging near point from the intercentral base line, varies somewhat with the age of the subject. One would suppose that in children, because of their high accommodation and smaller interaxial distance, the convergence distance would be much less than that in adults. As a matter of fact, it is not so greatly different. Thus, in 82 per cent of children under 16 and in 88 per cent of persons over this age it lies between 51 and 100 mm., which may thus be considered the usual range. The main difference between children and adults is that in the latter the proportion having a *PcB* over 90 mm. is greater and increases steadily with age, being 6 per cent in children, 12 per cent in adults under 40, 27 per cent in persons of from 41 to 45, 32 per cent in those between 46 and 50, and 40 per cent in those over 50. On the other hand, the proportion of persons with a *PcB* of less than 60, although very high in children (44 per cent), does not decrease much with age, being 20 per cent in adults below 45 and 17 per cent in those of 45 and over.

Except in persons over 50 a convergence distance of persistently over 110 is an evidence of an abnormally low converging power.

The foregoing facts are illustrated in the statistics in table 5, taken from my records.

In this series all cases were excluded which gave evidence of disturbing motor anomalies or of any great abnormality otherwise. In other words, the subjects may be regarded as fairly normal.[9]

In the summary in table 5 I have shown the values in meter-angles corresponding to the given convergence distance. These normally vary from 9 to 20; in children, from 10 to 22. From this it is evident how greatly the converging power exceeds its needs as age advances. A man of from 41 to 45 rarely, even with glasses, needs to converge closer than 25 cm. and thus never exerts more than 4 meter-angles of convergence. Yet 76 per cent of such men have a converging power of at least 15 meter-angles. Practically all have a converging power of 9 meter-angles, and, as stated, those who have a power much less than this (8 meter-angles or lower corresponding to a *PcB* of over 110) must, from their symptoms, usually be classed as abnormally deficient.

9. For further details see Duane, A.: The Norms of Convergence, in Contributions to Ophthalmic Science dedicated to Dr. Edward Jackson in honor of his seventieth birthday, Menasha, Wis., George Banta Publishing Co., 1926.

TABLE 5.—*Convergence Distance at Different Ages*

A. Number of Cases *

PcB	Age: Under 16	16-40	41-45	46-50	Over 50
40- 45	3	8	3	3	1
46- 50	10	9	1	5	1
51- 55	18	28	3	3	3
56- 60	19	46	5	7	5
61- 65	14	64	17	11	6
66- 70	12	43	5	8	1
71- 75	11	52	10	4	11
76- 80	10	35	6	16	8
81- 85	6	47	13	9	11
86- 90	4	26	9	7	3
91- 95	1	29	2	12	4
96-100	2	9	14	5	7
101-110	3	13	9	11	13
Over 110	1	0	2	6	9
	114	409	99	107	83

B. Percentages (Adults)

Convergence	Meter-Angles (Approximate)	Representing for a Mean Interaxial Distance of 62 Mm. a Convergence Angle of	Cases at Ages of: 16-40 No.	16-40 Per Cent	41-45 No.	41-45 Per Cent	46-50 No.	46-50 Per Cent	Over 50 No.	Over 50 Per Cent	All Over 40 No.	All Over 40 Per Cent
Very low (PcB over 110)	Less than 9	Under 26°	0	0	2	2	6	6	9	11	17	6
Low (PcB of 86-110)	9-12	16-20°	77	19	34	34	35	33	27	33	96	33
Medium (PcB 61-81)	12-16	21-27°	241	59	51	52	48	45	37	45	136	47
High (PcB 51-60)	16-20	28-32°	74	18	8	8	10	9	8	10	26	9
Very high (PcB 50-40)	20-25	32-38°	17	4	4	4	8	7	2	2	14	5
Total			409		99		107		83		289	

C. Percentages (Children)

Convergence	Meter-Angle	Representing for a Mean Interaxial Distance of 58 Mm. a Convergence Angle of	Cases: Number	Cases: Per Cent
Very low (PcB 100 or more)	10 or less	Under 16°	4	3
Low (PcB 81-100)	10-12	16-20°	13	11
Medium (PcB 56-80)	12-18	21-27°	66	58
High (PcB 46-55)	18-22	28-32°	28	25
Very high (PcB 45 or less)	22-26	32-38°	3	3
Total			114	

* In column one is given the PcB, i. e., the distance in millimeters of the convergence near point from the intercentral base line. In the succeeding columns is given the corresponding number of cases at the ages indicated.

Usual Values of the Convergence Angle.—If one knows the convergence distance (*PcB*) and the interaxial distance, one can determine the convergence angle from table 4, and thus arrive at a proper measurement of the converging power.

The determinations in table 6, made from a series of my own patients, in whom the relations were apparently normal, give a fair indication of the usual values.

For adults these figures agree fairly well with the statistics given in table 5 B. They indicate that the maximum convergence angle usually ranges from 16 to 32°. In nearly 60 per cent of subjects between 16 and 40 years of age it is between 21 and 27°. In presbyopes it tends to be less, in 40 per cent or more being below 21°. However, in persons under 45 a convergence angle of less than 16° is rare, and even in older subjects is uncommon.

TABLE 6.—*Maximum Convergence Angle at Different Ages*

	Age					
	Under 16		16-40		Over 40	
Convergence Angle	Number	Per Cent	Number	Per Cent	Number	Per Cent
Very low (under 16°)	0	0	0	0	2	3
Low (16-20°)	9	19	36	25	34	44
Medium (21-27°)	20	44	83	57	25	33
High (28-32°)	16	35	24	16	12	16
Very high (33-38°)	1	2	3	2	3	4
Total	46		146		76	

The probability, in fact, is that the actual converging power does not diminish greatly with age. Those who are old usually find it rather more difficult to exert converging power when called on to do so, simply because they are out of practice, having for years had no occasion to converge as young persons do. With practice, no doubt, most of them could learn to converge almost as well as ever.

That this is the case seems proved by the comparatively large number of cases in which even in presbyopes the converging power was found to be high. According to the different estimates given in tables 5 B and 6, a convergence angle of from 28 to 32° was found in from 16 to 18 per cent of adults below 40 and in from 9 to 16 per cent of those above 40, and it was very high, i. e., above 32° in from 2 to 4 per cent of both the younger and the older adults.

The statistics for children are inconclusive because of the small number examined. They indicate, however, that here also the convergence angle ranges regularly from 16 to 32°, but on the whole is rather higher than in adults, being over 20° in at least 80 per cent of the cases and over 28° in perhaps 30 or 35 per cent.

I may summarize these facts by saying that the maximum convergence adduction at all ages ranges generally from 16 to 32°, being thus about one half of the conjugate adduction, or the ability of the eye to rotate inward when performing a parallel movement. Except in persons above 50 a convergence adduction of less than 16° is unusual and probably abnormal, indicating a convergence insufficiency. This is made probable not only by the infrequency of this low range in normal subjects, but by the fact that when such a low range is persistently present it tends to be associated with symptoms indicative of motor trouble.

The highest maximum convergence angles that I found were 38° in a boy of 13 and one of 36° in a man over 50. In some cases, no doubt, these limits may be exceeded. Le Conte, for example, found that he himself had a convergence angle of 45°. Such a converging power is altogether exceptional. Moreover, measurements that indicate a high converging power are subject to many uncertainties. An error of 1 or 2 mm. in the measurement itself makes a considerable difference in the result, and moreover when the eyes are so excessively converged they probably no longer rotate about a fixed center, so that calculations of the actual angle of convergence are somewhat indeterminate.

Measurement of Convergence with Prisms.—Another method of determining the power of convergence is by ascertaining the prism convergence, or strength of prism, which can be overcome by the eyes when placed before the latter with its base out or toward the temple.

This method is analogous to that employed by Donders for determining the positive portion of the range of accommodation. The strength of prism overcome, in fact, represents the amount of residual convergence [10] that the subject under examination can exercise when his eyes are adjusted for the distance of the test object employed, just as the strength of the concave glass that he can overcome represents the amount of his residual accommodation under the same conditions. The amount of this residual convergence naturally varies with the distance of the test object, decreasing as the latter is brought nearer. It also varies considerably for the same distance in different persons, until the latter have by training learned to do what at the outset is quite difficult for them, namely, to look at a distant object and at the same time direct their eyes as if it were much nearer than it really is. Before this art has been learned, it will be found that the majority of persons cannot overcome prisms of over from 12 to 15°, base out, when looking at a distant object. Later, by constant practice, most persons get the ability to overcome prisms of from 60 to 70° or even 100° (equivalent to a convergence adduction of from 20 to 31° in each eye). One patient that I saw could cover a total of 120°, representing a convergence adduction of at least 35°. Thus, the maximum convergence

10. This is often improperly called the adduction.

produced in this way equals that produced in the natural fashion, i. e., by looking at a very near object.

Because of the difficulty that, as noted, most persons experience in overcoming prisms offhand and the great differences that subjects with evidently equal converging power show when thus tested, this method is an unreliable means of measuring the converging power. Its uncertainty is enhanced by the fact that the deviating effect of strong prisms alters a good deal with the way in which the prisms are held. Thus, if a prism of 50°, base out, is placed before each eye, the resulting deviation that each eye has to overcome may range from a minimum of 31 to 38° or more.

Convergence produced by prisms is at first associated with an accommodative effort similar to, but less than, that accompanying a natural convergence of the same degree. Thus, two cases that I examined showed the following amount of accommodation:

	Actual Amount of Convergence Produced by Overcoming the Prism	Accommodation Exerted	Corresponding Amount of Accommodation for a Natural Convergence of the Same Degree
Case 1	4.3°	0.25	1.25
	6.5°	0.50	2.00
	11.00°	1.50	3.25
Case 2	15.5°	2.50	4.50
	19.3°	5.00	5.50

By continual practice, however, the patient may learn to relax the accommodation while maintaining the convergence, and in this way prisms of 20 or 30°, base out, may be overcome without the accommodation being used at all. I have met with an extreme instance of this sort in which the patient could, without making any accommodative effort whatever, overcome prisms representing a convergence adduction of nearly 20° in each eye.

Divergence.—Divergence, or the simultaneous lateral separation of the visual lines, is a process that ordinarily subserves binocular fixation, being used when the eyes fix in succession objects more and more remote. The process may, however, be performed to excess, so that the visual lines diverge from the object of fixation, as when the homonymous diplopia caused by a prism placed base in before the eye is overcome, or, on the other hand, an involuntary or voluntary crossed diplopia is produced by turning the eyes outward.

Divergence of the sort last mentioned, i. e., that giving rise to a crossed diplopia, varies greatly in amount, and, although regarded as normal by those experimenters who have acquired a peculiar facility in producing it, is probably to be classed among the abnormalities. At all events, there are not many in whom the phenomenon is habitual, or who can produce it at will, and when present, it is generally associated with lack of muscular balance and other evidences of a pathologic state.

On the other hand, prism divergence,[11] or divergence produced in the act of overcoming a prism placed, base in, before the eyes, is an

11. This is usually but improperly called the abduction.

entirely normal phenomenon of definite character. Its maximum amount naturally varies with the distance of the object of fixation, increasing as the latter approaches the eye. The strength, in fact, of the prism, base in, that the eyes can overcome when regarding an object at any given distance represents the amount by which the eyes, when converged on the object and accommodated for the latter, can diverge, just as the strength of the convex glass that can be overcome in looking at the object represents the negative portion of the range of accommodation for the same distance.

In performing divergence both eyes turn out equally. It may seem as if they did not do so, e. g., when a prism, base in, is placed before one eye and the eye behind it turns out while the other stands still. But here, as in the case of the convergence produced by a prism, base out, it is a question of a conjugate movement superadded to one of divergence and tending to neutralize the outward movement of one eye and reenforce it in the other.

Divergence, in fact, is often thus associated with a conjugate movement, e. g., when one is overcoming a homonymous diplopia.

By turning both eyes out in thus overcoming prisms (and also voluntarily), the visual lines may be made to diverge a little beyond absolute parallelism. This constitutes an ultimate reserve of diverging power which it is important to possess, since, if it is absent, there is a tendency to homonymous diplopia and troublesome eye-strain in looking at distant objects. For far distance, indeed, the amount of this absolute divergence ranges in normal cases from 1 to 3.5° (representing an ability to overcome prisms, base in, of from 2 to 7▽). For the usual testing distance of 6 meters, this means a diverging capacity of from 1.5 to 4° (ability to overcome prisms, base in, of from 3 to 7.5▽). These may be regarded as the extreme limits, below or much above which the diverging power must be regarded as abnormal, but much the more usual for the diverging power for 6 meters is from 2 to 3° (representing a prism divergence of from 4 to 6▽).

Sherrington's Law as Applied to Divergence and Convergence; Essential Nature of These Two Functions.—There appears to me no reason to doubt that Sherrington's law, which governs both the movements of each eye separately and the conjugate movements of the two eyes, also governs convergence and divergence. If this is true, convergence consists in an equal simultaneous contraction of both interni combined with a simultaneous relaxation of both externi. Divergence would consist in exactly the reverse process.

Furthermore, the statements regarding the varying effectiveness in action of the muscles in moving the eye first outward and then back to the midline apply also to convergence and divergence. That is, the

muscles that effect either action work to the greatest advantage at the beginning of the movement and less and less advantageously as the movement proceeds. So when the eyes are diverged to their fullest extent (to or beyond parallelism) they converge most readily, and when they are converged to the extreme limit they diverge most readily. Also, as in the case of conjugate movements, the action of the main convergers (the interni) and the main divergers (the externi) is supplemented by accessory muscles, which make the variations in the movement less extreme and ensure smoothness of action.

Convergence is thus effected as follows:

The two externi are simultaneously inhibited and the two interni receive a simultaneous impulse to contract. At the same time the four elevators and the four depressors are called into play. The externi, being already shortened, are in position when relaxed to lengthen out readily. The interni, being on the stretch, can contract to the greatest advantage. The eyes thus would tend to move inward unduly fast if it were not for the brake action of the contracting obliques, which work as abductors. As the eyes turn well in and the relaxing externi and contracting interni are in less favorable mechanical conditions for furthering the movement, the obliques which previously hindered it cease their obstruction, since they no longer act as abductors, and the superior and inferior recti, now acting with increasing effectiveness as adductors, reenforce the in-turning of the two eyes. The four elevators and the four depressors furthermore, by their opposing vertical and torsion actions, keep the two eyes steady, preventing either one from swerving up or down.

Accordingly, while the effectiveness of the converging action is greatest at the outset of the movement and becomes less and less as it proceeds, the variations due to the increasing inefficiency of the lateral rotators are partially offset by the contraction of the elevators and depressors, which become increasingly effective in promoting convergence as the movement goes on. In extreme convergence movements, indeed, the superior and inferior recti probably take quite an important part.

Quite similar considerations apply to divergence, in which the four elevators and the four depressors act to reenforce and steady the movement. In passing from extreme convergence to parallelism or to actual divergence of the eyes, the action of the lateral rotators is effective at the outset, but is partly checked by the superior and inferior recti, which are still effective as adductors, and is not aided by the obliques, which in this position of the eyes do not act as abductors. Later on in the movement, when the actions of the lateral rotators is decreasingly effective in producing divergence, the obliques begin to help by their

increasing abducting action, and the superior and inferior recti lose their obstructive effect, since they begin to cease acting as adductors.

Many do not agree with the view here presented, that divergence is due in part to contraction of the externi and that it is a substantive function quite as much as convergence is. These objectors maintain that divergence is simply a relaxation of the interni, allowing the eyes to return to a position of rest.

Most observers who adopt the latter view assume that the natural position of the eyes, i.e., that in which all the muscles are fully relaxed, is one of slight divergence, parallelism itself requiring a certain tonic and constant contraction of the interni for its maintenance (Hansen Grut). Some, e. g., LeConte, have even thought that the position of complete relaxation is that in which each visual axis coincides with the axis of the orbit, a state of things implying a divergence of 45°. Those who think thus, however, appear to be misled in regarding as natural a condition that is abnormal, not to say pathologic. Schweigger argued strenuously against Hansen Grut's theory, and especially his application of it as explaining the nature of divergent squint, and Schneller also adduced a variety of arguments, which, however, are not convincing, to prove that the function of divergence is an active process. For my own part, I believe that in the majority of cases the position of perfect physiologic rest, if there is such a thing, is not one of divergence and that, in any event, the lateral separation of the visual lines must be regarded, in part at least, as an active process due to simultaneous contraction of the externi combined with an inhibition of the interni. One argument in favor of this is that many people, when their divergence is tested with prisms, experience a marked sense of strain analogous to that felt in overcoming prisms by converging the eyes. The latter is certainly an active process, and the former, therefore, in these cases at least, would seem to be one also. Patients, to be sure, who can diverge at will so as to produce crossed diplopia, often assure one that they do so by "relaxing" the eyes; but several observations have convinced me that this relaxation is really a muscular contraction.[12] Another argument that should have much weight is that elsewhere throughout the body wherever a voluntary movement in one direction is replaced by a movement in the contrary sense the change is regularly effected not simply by a relaxation of the muscle that has been contracted, but also mainly by the active contraction of a muscle opposing it. The view that the external recti thus take an active part in the diverging process is furthered by the experiments of Sherrington, which showed that an inhibition of these muscles was able to produce a moderate convergence even when all the other ocular muscles, including, of course, the internal recti, were divided. If the external recti are subject to a central inhibition so as to effect some degree of convergence, they should also be subject to a central stimulation that would effect divergence.

But whether produced by passive inhibition or by active stimulation or, as I believe, by both, divergence is evidently a substantive physiologic process for which some distinct nervous mechanism is provided. Pathologic facts make this evident.

12. A similar instance in which an undoubted muscular contraction was described by the patient as a "relaxation" was one that I met with, in which a homonymous diplopia of 8° (15▽) was produced at will. Here, of course, a condition of convergence was present, which could have been brought about only by an active contraction of the interni.

Divergence in a Vertical Plane.—Divergence in a vertical plane (supervergence,[13] the see-saw movement of Maddox) is a simultaneous and equal elevation of one eye and depression of the other. Supervergence is called right or left, according as the right or the left eye is the higher.

As a rule, supervergence is done only in the interests of binocular single vision, i. e., to secure fusion when the images of the two eyes are put on different levels either by prisms or by some deviation. Some persons can perform this movement with the opposite effect, i. e., so as to produce or exaggerate a vertical diplopia. In such persons the facility of supervergence may be rather considerable, so that they are able to produce or correct a vertical diplopia of 5° or more. Usually, however, the angle through which one can diverge the eyes in a vertical plane does not exceed 1° (corresponding to a 2$^{\nabla}$ prism, base up or down).

The contrary movement—convergence of the eyes in a vertical plane when they have been separated by a vertical divergence—also takes place. This is seen when a person who has overcome a prism, base down, before one eye suddenly removes the prism. For a moment he sees double on account of the vertical divergence of the visual lines. But this diplopia at once disappears, evidently because the two divergent visual lines once more settle by a vertically converging movement into a state of parallelism.

Formerly, when it was thought that only the eye behind the prism was concerned in overcoming the diplopia caused by the latter, the ability to overcome a prism placed base down before the right eye was called right sursumduction and the ability to overcome a prism placed base up was called right deorsumduction. However, as indicated from the foregoing statements, right deorsumduction is synonymous with left sursumduction, and with left supervergence. The term deorsumduction thus has no real meaning.

Convergent or Divergent Torsions.—The vertical meridians, instead of remaining parallel, may tip toward or away from each other. The former condition may be called contorsion and is associated with what I have called intorsional diplopia. A tipping apart of the vertical meridians may be called distorsion and is associated with extorsional diplopia. Distorsion occurs normally in positions of marked convergence, the vertical meridians then diverging at their upper extremities, and the amount of divergence increasing as the eyes are elevated (Meissner, Le Conte). Le Conte[14] found in his own case that when

13. It is also called sursumduction, but the term duction is regularly reserved for monocular movements, and sursumduction (or superduction) should mean simply elevation of one eye, a condition that would nearly always be associated with elevation (not depression) of the other eye.

14. Le Conte, J.: Sight: An Expression of the Principles of Monocular and Binocular Vision, New York, D. Appleton & Company, 1881, p. 184.

the two eyes were turned in so as to represent a convergence angle of 9° the extorsion of each eye was 1.25° and rose to 2.5°, 5° and 10° for a convergence angle of 14°, 22° and 25°, respectively. But, apart from the physiologic torsion movement, a divergent or convergent rotation of the vertical meridians may be produced in the normal eye and demonstrated by suitable apparatus (Helmholtz).

One must differentiate the physiologic torsions from the pathologic conditions in which the vertical meridians tilt toward or away from each other as a result of monocular paralyses and thus produce a corresponding diplopia.

A CENTRAL VISION SCOTOMETER

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In case there is no opacity of the media of the eye, low central vision may be due to two causes: a refractive error or a centrally located defect in the sensorium. The separation of these two causes presents a somewhat difficult problem in diagnosis. Among the possibilities available the following may be noted: (*a*) An attempt may be made to improve vision by corrective glasses. (*b*) The eye may be refracted by one or more of the objective methods. (*c*) The influence of refractive errors may be reduced to a minimum by the use of a very small artificial pupil in close proximity to the cornea. (*d*) With the best corrective glass that can be obtained, an examination may be made for a central scotoma.

In the instrument to be described, the following selection is made from these possibilities: The eye is rendered practically independent of refraction, and a means is provided for making a quick examination for a central scotoma and a rough determination of its size.

The instrument should be a valuable aid to the refractionist.[1] In cases of low acuity he can in a few minutes determine whether a central scotoma is present, and he is saved a futile attempt to bring about normal vision through refractive correction. The use of the instrument thus saves a great deal of valuable time and leads to a prompt reference of the case to the appropriate division of the clinic. Obviously, the instrument is also of great service in the study of the visual field. The mapping, and even the detection of small central scotomas, is one of the outstanding problems in scotometry. With this simple device, the detection of the scotoma and the assignment to its proper place in a graduated scale of sizes can be made in a few minutes. In some cases we have been able to detect scotomas with the instrument which we failed to discover in a careful study of the central field with the tangent screen, and in all cases we have been able to make the examination in a small fraction of the time required for the study with the tangent screen.

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1. From our own experience with the instrument, we cannot speak too highly of its usefulness in a refraction clinic for the type of patients who come to such a clinic in a hospital for ocular diseases.

Further, in no case have we ever failed to discover with the instrument a scotoma that has been disclosed by the most careful and meticulous use of the tangent screen.

It is not to be understood, however, that the instrument can be used in this respect as a substitute for the tangent screen. If a detailed outline of the scotoma is wanted, the type of method used on the tangent screen has to be employed. For all cases in which it is sufficient to know that a scotoma is present and to be informed as to its density and approximate size, the instrument is adequate. It also is extremely valuable for quick examinations preliminary to more detailed study with the tangent screen.

ADVANTAGES AND GENERAL PLAN OF INSTRUMENT

The features and advantages of the instrument may be summarized briefly as follows:

1. The determination can be made in but a small fraction of the time required for an examination with the tangent screen. With the eye in position, no more time is required than to look at the stimulus presented. The ease and quickness with which the examination can be made are of particularly great advantage in cases in which the patient is too ill for an examination with the tangent screen.

2. Sensitive means are provided for the detection of scotomas of different degrees of density, and also for a determination of their approximate size. Colored stimuli, which are graded as to size, and form stimuli, which are graded as to both size and relation to background, are used.

3. The determinations are made with the correct control of preexposure and the surrounding field. For colored stimuli, the preexposure and surrounding field are of a gray, the brightness of the color in central vision for the intensity of illumination and the size of the pupil used, and for the form stimuli, the preexposure is of the brightness of the surrounding field.

4. Convenient mechanical means are provided for the control of both the preexposure and the length of exposure, and also for applying an objective check on the correctness of the judgment.

5. The test surfaces are uniformly illuminated with light corrected to daylight color. Means are provided for varying the intensity of illumination. However, the effective illumination can be held constant at 7 foot-candles, as is done in the Ferree-Rand perimeter, if that is desired.

6. The determination is rendered practically independent of the condition of refraction, without the inconvenience and disturbance attending the wearing of correcting glasses. In an examination for

central scotoma, the elimination of the influence of refractive condition is of great importance.

7. Fixation is controlled by the vision of the eye under examination. Reliance does not have to be placed on the control afforded by coordination with the fixation of the eye not under examination. In this connection it may be noted that the eye not under examination also frequently has a central scotoma.

8. A stationary stimulus with a controlled exposure is used. The errors attending the use of a moving stimulus are thus avoided.[2] It is extremely difficult to detect very small central scotomas on the tangent screen even with the best control of fixation and under the most favorable conditions of presentation of stimulus that can be obtained.

The general plan of the instrument is as follows:

1. The colors, whites, blacks and grays, which serve as stimuli and preexposure, are surfaced in sectors on a rotatable disk of suitable size, disk 1.

2. Immediately in front of this disk, displaced a suitable distance to one side, is a second rotatable disk, disk 2, which contains the apertures through which the stimulus surfaces are viewed. These apertures are graded in size and are appropriately spaced on the circumference of a circle about the center of rotation of the disk. The disk is divided into three equal sectors, each of which contains three stimulus apertures. The sectors are surfaced to give the brightness of the background or surrounding field (gray or black) which is required for each of the stimuli. As the disk is rotated the stimuli are thus presented to the eye in the desired relation to the background.

3. In front of these two disks is a stationary plate with a large circular aperture. This plate provides for the more remote control of the brightness of the surrounding field and contains the device for the control of fixation.

4. The foregoing devices are mounted at one end of a horizontal bar. At the other end, on an upright support, is the eyepiece, which is

2. The following errors attending the use of moving stimuli in the study of the field of vision may be noted: (*a*) A much poorer control of fixation is had however good a fixation device is used. That is, there is a tendency for the eye to follow the moving stimulus, the act of fixation is prolonged unduly, and the eye of the patient is not under the observation of the examiner at the time the judgment is made. (*b*) The variable effects of previous exposures of the eye on the sensitivity of the retina at the point of examination cannot be so effectively eliminated. (*c*) There is a time error which cannot be standardized. The report is given after the stimulus has passed the critical point, the amount of error depending on both the rate of movement of the stimulus and the slowness of the reaction of the patient. The reactions of many pathologic observers, it may be noted, are very slow.

provided with a pupillary aperture of the size which we have found to be of optimum service in eliminating errors of refraction and in giving a high acuity. The disks and the field plate are illuminated by two miniature lamps in small housings which are carefully light-proofed. These housings are provided with etched daylight filters for color correction and with neutral filters for gross control of intensity. They are mounted on a horizontal support, which may be moved backward and forward on the horizontal bar mentioned or clamped in position at any point desired. The position of the lamp houses on the transverse support has been chosen to give the best possible illumination of the test object compatible with a suitable angle of separation as viewed by the eye.

5. The instrument is mounted on a tripod stand. This stand is provided with a set screw adjustment for height.

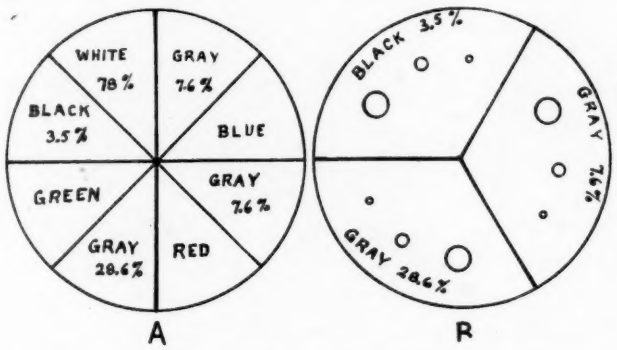

Fig. 1.—*A*, disk 1, which provides the stimuli and preexposure. *B*, disk 2, which provides the stimulus apertures and surrounding fields.

DETAILS OF CONSTRUCTION AND METHOD OF USING

Disk 1 is shown in figure 1 *A*. It is divided into eight equal sectors, which are surfaced with especially prepared paints and pigment papers in the following order: black (coefficient of reflection 3.5 per cent); white (coefficient of reflection 78 per cent); gray of the brightness of the green of the Heidelberg papers (coefficient of reflection 28.6 per cent); the green of the Heidelberg papers; gray of the approximate brightness of the blue of the Heidelberg papers (coefficient of reflection 7.6 per cent); the blue of the Heidelberg papers; gray of the approximate brightness of the red of the Heidelberg papers (coefficient of reflection 7.6 per cent), and the red of the Heidelberg papers. The grays are approximately of the brightness of the colors when viewed through the small aperture used in the eyepiece. Thus, when there is a scotoma for color the stimulus tends to disappear completely and the background to

be seen as uniformly gray.[3] The disk is arranged in the manner described, in order that when it is rotated the form and color stimuli selected for the examination will be presented in a given prescribed order, each preceded by its correct preexposure. That is, the white, green, blue and red sectors serve as stimuli; the grays, respectively, of the brightness of the green, blue and red stimuli, as preexposures for these stimuli, and the black as a preexposure for the white stimulus. Since these preexposure sectors are of the same brightness as the background for the stimuli, they may be substituted as blanks when desired to serve as an objective check on the correctness of the judgment.

Disk 2 is shown in figure 1 *B*. The three sectors of this disk are surfaced with gray of the approximate brightness of red and blue of the Heidelberg series of papers (coefficient of reflection 7.6 per cent); gray of the brightness of the green of the Heidelberg papers (coefficient of reflection 28.6 per cent), and black (coefficient of reflection 3.5 per cent). The diameters of the three apertures contained in each sector are, 1.4, 2.9 and 5.75 mm., respectively. The angles subtended at the eye, 33 cm. distant, are 0.25, 0.5 and 1 degree.

The stationary plate mentioned under 3 is surfaced with the darker of the two grays used for disks 1 and 2, the coefficient of reflection being 7.6 per cent. Centrally located in this plate is a circular aperture through which is seen the test object on its appropriate background. This plate also contains the device used for the control of fixation. This device consists of a circle, outlining the aperture, and two horizontal and two vertical arms, extending outward from the circle at diametrically opposite points, so located that their prolongation inward intersects at the center of the aperture. The device serves to guide and control fixation by establishing well marked lines of direction which intersect at the center of the test field. In making the examination the patient is instructed to look at the center of the circle at this point of intersection. When this is done, an effective control of fixation is secured even though a central scotoma is present.[4]

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3. No one gray can be found for which this is literally true for all observers. Observers, particularly pathologic ones, differ in their achromatic response to color. The grays selected are those which match the colors for the majority. For some, however, they will be slightly too light and for others too dark. Since this is the case and since the red and blue of the Heidelberg papers are so nearly the same in brightness for the conditions under which they are viewed, it was felt that one brightness of surrounding field and preexposure could serve for both colors. By employing the same gray for these two colors, disk 2 could be made with three instead of four sectors, which gives the instrument greater compactness and neatness of construction.

4. The effectiveness of this combination of circle and broken cross for the control of fixation of eyes having a central scotoma will be shown in a paper to be published by us in collaboration with L. L. Sloan.

As already indicated, the purpose of the first disk is to provide a conveniently operable device for the presentation of the color and form stimuli selected for the examination, the preexposures needed for these stimuli and the blank surfaces for an objective check on the judgment. The second disk provides the background on which the stimulus is to be viewed and the control of the brightness of the more immediately surrounding field, and contains the apertures needed for the gradation of the size of the stimulus. The stationary front or field plate provides control of the brightness of the more remote surrounding field and contains the device for the control of fixation.

By means of these devices it is possible: (1) to present stimuli of a great variety of visibilities, color and form, e. g., red, blue or green on a gray of the brightness of the color; white on a background of black, gray of the brightness of red and blue, or gray of the brightness of green; gray of the brightness of green on a background of black or gray of the brightness of red and blue, and gray of the brightness of red and blue on a background of black, or of gray of the brightness of green. Stimuli of a great variety of visibilities, color and form are thus made available and there is provided for each colored stimulus a background of the brightness of the color, and for each form stimulus, a background of black or of grays of the brightness of the colors.

(2) To provide for each colored stimulus a preexposure of the brightness of the color, and for each form stimulus, a preexposure of the brightness of the background, the sector serving as preexposure, for convenience of operation, being made to precede, in the order of rotation of the disk, the sector that serves as a stimulus.

(3) To present each stimulus in three sizes, 1.4, 2.9 and 5.75 mm. in diameter, and subtending at the eye angles of 0.25, 0.5 and 1 degree.

(4) To present at will, as the substitute for any stimulus, a neutral surface of the brightness of the surrounding field to serve as a blank for an objective check on the correctness of the judgment.

Figure 2 shows a front and a back view of the instrument. The eyepiece is in the form of a tube 28 mm. in diameter and 45 mm. long. The diaphragm forming the front of this eyepiece is made slightly concave to fit closely the anterior surface of the eye. At the center of this diaphragm is a circular aperture 1.16 mm. in diameter. In order to eliminate reflections and scattered light, the inside of the tube is painted black. The eyepiece is mounted on an upright support 45 mm. high at one end of the horizontal bar. At the other end, 33 cm. from the pupillary aperture, is the field plate. This plate is 11 cm. square. The central aperture is 17.3 mm. in diameter and has a flatly beveled edge. This aperture subtends an angle of 3 degrees at the eye. The beveled edge is 2 mm. in breadth. This edge, which is painted white to

give it high visibility, provides the circle which forms a part of the fixation device. The horizontal and vertical arms, which as a further aid to fixation extend outward from this circle in the form of a cross, are 17.4 mm. long and 2 mm. broad. The angles subtended by these two dimensions at the eye are 3 and 0.33 degrees, respectively. The total angle subtended by cross and aperture is 9 degrees. The arms of the cross are also painted white to give a high visibility.

Disk 1 and 2 are each 7 cm. in diameter and are made of thin sheet brass. They rotate about the shank of a very short screw, which is threaded into the back of the field plate. The centers of rotation of

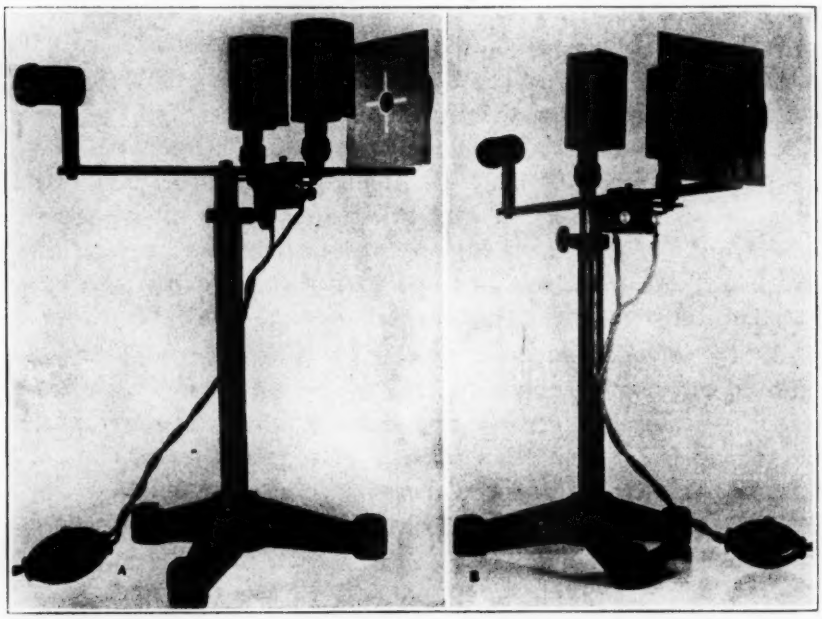

Fig. 2.—*A*, front view of the instrument, showing 0.5 white stimulus on a black background. Front plate illuminated. *B*, back view of the instrument.

these disks are 48 mm. apart, and each is 24 mm. from the center of the circular aperture in the field plate. The stimulus apertures in disk 2 are on the circumference of a circle the center of which is the center of rotation of the disk and the radius of which, 24 mm., is the distance between the center of rotation of the disk and the center of the circular aperture in the field plate. When in position in front of the eye, the center of each aperture coincides exactly with the center of the aperture in the field plate. In order that each aperture will automatically take this position in turn when the disk is rotated, nine stops are provided. These stops are constructed as follows: The back wall of the disk contains nine shallow cups, suitably spaced on a circle of a radius of 14 mm.,

10 mm. less than the radius of the circle containing the centers of the nine apertures. At the proper level to engage the cups as they pass, a small ball or bead is soldered to the front surface of a steel lamina near its upper end. The lower end of this lamina is fastened to the back surface of the field plate. Acting as a spring, this lamina gives the tension that is needed to stop the disk when the ball engages a cup.

The positioning of the ball and the spacing of the cups have, of course, to be made with great accuracy and precision if the center of each aperture is to come to rest at the exact center of the aperture in the field plate. A similar stop device is provided for disk 1, so arranged that the disk automatically comes to rest when the radius bisecting any one of the stimulus sectors cuts the center of the aperture of the field plate. For convenience of rotation each disk is provided with a milled edge, a small portion of which projects beyond the edge of the field plate, and to permit of smoothness and freedom of movement in turning, the circles containing the cups are grooved. With the provisions made for convenience and accuracy of adjustment, the stimuli, backgrounds and preexposures can be quickly and easily changed without attracting the attention of the patient. In order to guard against any trace of shadowing of the surface exposed, the disks are made of very thin sheet metal, no. 21 B. & S. gage, and the edges of all the apertures are beveled as much as the thickness of the metal will permit. With the exception of the stem, the tripod base and the two disks the instrument is painted gray of the same coefficient of reflection as the field plate.

The instrument is inexpensive, requires a very small amount of space and is exceedingly simple and easy to operate. Little skill is demanded either of the examiner or of the patient in making the examination. With the eye in position at the eyepiece, the smallest aperture in the sector of the gray required for the background or surrounding field on disk 2 is turned into position at the center of the opening in the front stationary plate. The sector of disk 1 required for preexposure is then rotated into position behind this aperture. The fixation is taken, and at a signal given by the examiner the stimulus sector, which in every case is immediately adjacent to the preexposure sector, is turned into position behind the aperture, and the patient is required to report what is seen. In order to provide an objective check on the correctness of the judgment, the preexposure may be alternated with the stimulus sector. When this is done, there should be no difference in appearance between the aperture and the surrounding field. If the patient fails to discriminate the stimulus with this size of aperture, the examination is repeated with the larger sizes. If the stimulus is seen with either of these two sizes of aperture, one of two conclu-

sions may be drawn: (*a*) The scotoma is smaller than the stimulus seen, or (*b*) the stimulus is of a degree of visibility which is suprathreshold for the defective area. Which of these conclusions is correct can in many cases be ascertained by questioning the patient. In the former case, only a rim (positive scotoma) or all the stimulus (negative scotoma) is seen as of normal visibility or nearly so; in the latter, the stimulus appears of reduced visibility.

The instrument may be recommended also for the delicacy and accuracy with which the examination can be made. Not only have we as yet never failed to discover with it any scotoma that was detected with the tangent screen, but in several cases we have detected scotomas which were missed in ordinarily careful examination with the tangent screen. In all these cases the finding with the scotometer was, it may be noted, later verified by a more careful examination with the tangent screen. It is our belief that when an equal amount of care is exercised in the use of both instruments, the scotometer has greater sensitivity for the detection of small central scotomas than has the tangent screen. Its use in the refraction clinic will, as already stated, save a great deal of time and secure a correct reference of the case when a central scotoma is present. Its small cost, convenience of size and simplicity and quickness of use especially fit it for this type of service. In addition, it should constitute an almost indispensable part of the equipment for the routine and general examination for ocular diseases.

CHEMISTRY OF THE LENS

II. COMPOSITION OF BETA CRYSTALLIN, ALBUMIN (GAMMA CRYSTALLIN) AND CAPSULE

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In the first paper on the chemistry of the lens,[1] the analyses of the albuminoid and alpha crystallin were given. The present paper continues with the report on the analyses of the beta crystallin, albumin (gamma crystallin) and capsule of the lens. The last two proteins occur in such small quantities in the lens that the material from many thousands of eyes was allowed to accumulate in order to obtain sufficient protein for repeated analyses.

Of the three proteins, beta crystallin has been most completely studied in the past. Mörner found 17.04 per cent of nitrogen and 1.27 per cent of sulphur in beta crystallin,[2] and 14.1 per cent of nitrogen and 0.83 per cent of sulphur in capsule.[3] Jess, disregarding the presence of albumin, reported 17 per cent of nitrogen and 1.34 per cent of sulphur in beta crystallin.[4] By the use of a method of isolation of amino-acids, he estimated that beta crystallin consisted of 4.6 per cent of lysine, 2.49 per cent of histidine, 7.54 per cent of arginine,[5] 4.9 per cent of cystine, 3.7 per cent of tyrosine and an undetermined percentage of tryptophan.[6] Jess isolated various amounts of other amino-acids, which need not be mentioned here. Mörner reported on only the incomplete elementary analysis of the albumin.

The methods for the preparation and purification of the beta crystallin and albumin have been published.[1] The lens capsule was

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From the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

1. Krause, A. C.: Chemistry of the Lens: I. Composition of Albuminoid and Alpha Crystallin, Arch. Ophth. **8**:166 (Aug.) 1932.

2. Mörner, C. T.: Untersuchungen der Proteïnsubstanzen in den leichtbrechenden Medien des Auges: I, Ztschr. f. physiol. Chem. **18**:61, 1894.

3. Mörner, C. T.: Untersuchungen der Proteïnsubstanzen in den leichtbrechenden Medien des Auges: III, Ztschr. f. physiol. Chem. **18**:233, 1894.

4. Jess, A.: Die Monoaminosäuren der Linsenproteine, Ztschr. f. Physiol. Chem. **110**:266, 1920.

5. Jess, A.: Der Gehalt der Linsenproteine an Histidin, Arginin und Lysin, Ztschr. f. physiol. Chem. **122**:160, 1922.

6. Jess, A.: Die moderne Eiweisschemie im Dienste der Staorforschung: I, Arch. f. Ophth. **105**:428, 1921.

easily separated from most of the lens substance. The capsule was then carefully washed free of lens substance, epithelium and zonular fibers. Occasionally, a sample capsule was studied microscopically, after staining, to determine the efficiency of the washing. The capsules were washed with distilled water, alcohol and then ether, and were dried at 105 C.

TABLE 1.—*Elementary Composition of Proteins* *

Preparation	Carbon, per Cent	Hydrogen, per Cent	Nitrogen, per Cent	Sulphur, per Cent	Ash, per Cent
Beta crystallin	53.59	6.88	17.05	1.32	0.90
				1.35	
	53.54	6.93	17.04	1.26	0.91
Average	53.57	6.91	17.05	1.31	0.91
Albumin	53.66	6.72	17.43	0.91	0.99
	53.71	6.73	17.26	0.93	0.95
Average	53.69	6.73	17.35	0.92	0.97
Capsule	46.98	6.93	14.07	0.85	0.93
	47.18	7.09	14.02	0.83	0.95
Average	47.08	7.02	14.05	0.84	0.94

* The percentages are calculated on an ash-free and moisture-free basis. Carbon and hydrogen are estimated by Pregl's method.

TABLE 2.—*Distribution of Nitrogen in Beta Crystallin as Determined by the Van Slyke Method* *

	Preparation				
Nitrogen	I,† Gm.	II,‡ Gm.	I, per Cent	II, per Cent	Average, per Cent
Total	0.3135	0.4687	16.89	17.01	16.95
Amide	0.0219	0.0312	7.02	6.85	6.93
Humic acid	0.0053	0.0084	1.69	1.80	1.74
Cystine§	0.0097	0.0144	3.09	3.09	3.09
Arginine	0.0682	0.1018	21.75	21.51	21.63
Histidine	0.0212	0.0324	6.76	6.90	6.83
Lysine	0.0164	0.0242	5.23	5.16	5.20
Amino-nitrogen of filtrate	0.1535	0.2267	48.96	48.37	48.66
Nonamino-nitrogen of filtrate	0.0202	0.0307	6.44	6.55	6.49
Recovered	0.3164	0.4698	100.90	100.23	

* The figures for nitrogen are corrected for the solubilities of the phosphotungstates of the basic amino-acids. The percentages are calculated on an ash-free and water-free basis.

† 1.8554 Gm. of protein.

‡ 2.7554 Gm. of protein.

§ Calculated from sulphur.

TABLE 3.—*Distribution of Nitrogen in Albumin (Gamma Crystallin) of the Lens as Determined by the Van Slyke Method* *

Nitrogen	Preparation I,† Gm.	Preparation II,‡ Gm.	I, per Cent	II, per Cent	Average, per Cent
Total	0.1698	0.2230	15.76	15.76	15.76
Amide	0.0117	0.0154	6.88	6.91	6.90
Humic acid	0.0032	0.0032	1.88	1.43	1.65
Cystine§	0.0045	0.0059	2.64	2.64	2.64
Arginine	0.0442	0.0582	26.03	26.11	26.07
Histidine	0.0097	0.0128	5.70	5.74	5.72
Lysine	0.0109	0.0141	6.41	6.32	6.37
Amino-nitrogen of filtrate	0.0687	0.0926	40.46	41.52	40.99
Nonamino-nitrogen of filtrate	0.0172	0.0214	10.13	9.59	9.86
Recovered	0.1701	0.2236	100.13	100.14	

* The figures for nitrogen are corrected for the solubilities of the phosphotungstates of the basic amino-acids.
† 1.0757 Gm. of protein.
‡ 1.4154 Gm. of protein.
§ Calculated from sulphur.

TABLE 4.—*Distribution of Nitrogen in Lens Capsule as Determined by the Van Slyke Method* *

Nitrogen	Preparation I,† Gm.	Preparation II,‡ Gm.	I, per Cent	II, per Cent	Average, per Cent
Total	0.1698	0.2570	14.31	14.30	14.31
Amide	0.0086	0.0129	5.06	5.01	5.03
Total humic acid	0.0029	0.0048	1.70	1.87	1.79
Cystine§	0.0045	0.0068	2.61	2.61	2.61
Arginine	0.0256	0.0390	15.08	15.17	15.13
Histidine	0.0074	0.0120	4.35	4.66	4.51
Lysine	0.0095	0.0141	5.59	5.49	5.54
Amino-nitrogen of filtrate	0.0961	0.1406	56.59	54.51	55.55
Nonamino-nitrogen of filtrate	0.0187	0.0280	11.01	10.93	10.97
Recovered	0.1733	0.2582	101.99	100.25	

* The figures for nitrogen are corrected for the solubilities of the phosphotungstates of the basic amino-acids. The percentages are calculated on an ash-free and water-free basis.
† 1.1873 Gm. of protein.
‡ 1.7966 Gm. of protein.
§ Calculated from sulphur.

TABLE 5.—*Distribution of Nitrogen in Beta Crystallin, Albumin (Gamma Crystallin) and Lens Capsule as Calculated from the Van Slyke Analysis in Terms of Percentage of the Proteins*

Nitrogen	Protein: Beta, Average Percentage	Albumin, Average Percentage	Capsule, Average Percentage
Amide	1.13	1.08	0.72
Humic acid	0.26	0.26	0.25
Basic	6.24	6.43	3.98
Nonbasic	9.35	8.02	9.53
Total	16.99	15.79	14.47

ANALYSES OF THE PROTEINS BY THE VAN SLYKE METHOD [7]

Duplicate samples of about 2 Gm. of each of the proteins were hydrolyzed by boiling for thirty hours with 150 cc. of 20 per cent hydrochloric acid. The phosphotungstates of the bases were decomposed by the amyl alcohol-ether method.[8] The distribution of nitrogen in the beta crystallin, albumin and capsule is shown in tables 2, 3, 4 and 5. The amino-acids in the protein as calculated from the Van Slyke and colorimetric analyses are given in table 6.

DETERMINATION OF TYROSINE, TRYPTOPHAN AND CYSTINE

The determination of tyrosine and tryptophan was done according to the colorimetric method of Folin and Marenzi.[9] In addition to the

TABLE 6.—*Basic Amino-Acids in Beta Crystallin, Albumin (Gamma Crystallin) and Lens Capsule* *

Amino-Acid	Protein		
	Beta, Average Percentage	Albumin, Average Percentage	Capsule, Average Percentage
Arginine †	11.44	12.80	6.73
Histidine †	4.28	3.32	2.32
Lysine †	4.61	5.30	4.15
Cystine ‡	4.47	3.58	3.25
Cystine §	4.30	1.83	1.48
Cystine #	3.18		
Tyrosine ‖	5.47	4.19	4.98
Tryptophan ‖	1.37	0.88	0.58

* Calculated on an ash-free and water-free protein.
† The percentages were calculated from the average results of duplicate analyses by the Van Slyke method.
‡ See †. Not to be considered as actual cystine, but as an arbitrary value derived from the amount of sulphur.
§ Determined by the Folin and Marenzi method as modified by Tompsett.
# Determined by the Sullivan method.
‖ Determined by the Folin and Marenzi method.

values found for cystine by the Van Slyke method, there are also included in table 6 figures for this amino-acid obtained colorimetrically by the methods of Sullivan [10] and Tompsett.[11] The method of Van

7. Van Slyke, D. D.: The Analysis of Protein by Determination of the Chemical Group Characteristics of the Different Amino Acids, J. Biol. Chem. **10**: 15, 1911-1912. Koehler, A. E.: Modification of the Van Slyke Method for Determining Arginine, J. Biol. Chem. **42**:267, 1920.

8. Van Slyke, D. D.: Improvements in the Method for Analysis of Proteins by Determination of Chemical Group Characteristics of Different Amino Acids, J. Biol. Chem. **22**:281, 1915.

9. Folin, O., and Marenzi, A. D.: Tyrosine and Tryptophane Determinations in One-Tenth Gram of Protein, J. Biol. Chem. **83**:89, 1929.

10. Sullivan, M. X.: A Distinctive Test for Cystine, Pub. Health Rep. **41**: 1030, 1926; The Colorimetric Estimation on Cystine in Casein by Means of the Naphthoquinone Cysteine Reaction, J. Biol. Chem. (Proc. Soc. Biol. Chem.) **74**:14, 1927.

11. Tompsett, S. L.: A Note on the Determination of Cystine in Proteins by the Method of Folin and Marenzi, Biochem. J. **25**:2014, 1931.

Slyke gives arbitrary values for cystine, while the method of Sullivan is highly specific and is considered to give the better results.

COMMENT

The analyses of the crystallins show definitely that they differ from one another and from serum globulins. The calculated percentages of arginine, histidine, lysine and tyrosine present in beta crystallin are naturally greater than those found by Jess, who used the isolation method. The values obtained with the Van Slyke method, although they are arbitrary, are probably more correct than those of Jess. Values obtained by the use of the present improved methods for the isolation of arginine, histidine and lysine are needed to check the percentages of these amino-acids; unfortunately, however, the recent methods of isolation require excessively large quantities of protein.

It is interesting to note that Tsuji,[12] from analyses of the proteins of experimentally induced cataracts, found that in the cataracts the total amount of proteins and cysteine decreased, and the water and the ratio of the insoluble to the soluble proteins increased. According to the data, the beta crystallin evidently gradually became insoluble through a "denaturation" partly caused by the loss of sulphydryl groups. Moreover, the same reaction was found to occur in this laboratory, when beta crystallin was kept in solution at room temperature for any length of time. The amount of sulphur in the beta crystallin decreased rapidly until about one third of it was lost and the protein was denatured.

The albumin of Mörner (or gamma crystallin of Woods) has not been previously characterized chemically. The Van Slyke analyses indicate that it falls into the general class of albumins. It is noteworthy that the amount of cystine in albumin approaches that in beta crystallin, yet it is perhaps too early to suggest that this cystine in albumin plays a part in the internal metabolism of the lens similar to that played by the cystine in beta crystallin. It is quite true that the more closely a protein is associated with cell proliferation, the greater is its content of diamino-acids, particularly arginine. Therefore, it is remarkable that albumin and beta crystallin contain more of these acids than albuminoid does.

Contrary to the ophthalmologic reports and reviews, it must be strongly emphasized that no glutathione has ever been isolated from any lens protein or any other protein, but that it has been found in the free state in the lens and other tissues. The cystine of the lens proteins may be partly derived from the cysteine of the protein molecule. Likewise, however, cysteine that has been isolated from protein hydrolysates has never been proved to be the cysteine of the protein molecule.

12. Tsuji, T.: Experimentelle Untersuchungen über das Linseneiweiss bei Katarakt, J. Biochem. **15**:33, 1932.

The evidence that cysteine is a part of the molecule is indirect and circumstantial, since it is based on the nitroprusside, iodine and lead acetate reactions. It must be remembered that the sulphydryl is readily oxidized by air, and that the humic acid formed during hydrolysis contains cystine or its decomposition products,[13] and, therefore, the quantitative analysis or isolation methods give only approximate minimal values. Any iodine method that is used for the estimation of either cystine or cysteine in hydrolysates of lens proteins may be criticized,[12] in spite of its extensive use.[14] The iodine method is incapable of differentiating cystine from other disulphides.[15] However, in any case, the cystine or cysteine calculated as cystine does not account for all the sulphur. It is possible that in the lens proteins, the remaining sulphur is in the form of methionine.

Because of the chemical and physical characteristics of the four proteins in the lens substance, there is no need to change the names which Mörner applied to these proteins. No two proteins of the lens substance are chemically identical. Although through physicochemical manipulation a lens protein may acquire somewhat the physical-chemical properties of another lens protein, up to the present time there is no reason in the least to suppose that one of these proteins may change its chemical structure to that of another lens protein.

The protein of the capsule has not been previously studied chemically except for a partial elementary analysis. In many ways it is an unusual protein. The large amount of oxygen in the molecule together with nitrogen is suggestive of an amino-carbohydrate. Further evidence is the quantitative carbohydrate reaction, which shows a dextrose equivalent of approximately 10 per cent. On the other hand, the albuminoid, alpha crystallin, beta crystallin and albumin of the lens show only about a 0.1 per cent dextrose equivalent of carbohydrate when tested according to the method of Tillmans and Philippi [16] or Dische and Popper.[17]

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13. Roxas, M. L.: The Reaction Between Amino Acids and Carbohydrates as a Probable Cause of Humin Formation, J. Biol. Chem. **27**:71, 1916.

14. Shinohara, K.: Oxidation of Cystine by Iodine in Aqueous Medium, J. Biol. Chem. **96**:285, 1932. Andrews, J. C.: The Oxidation of Cystine in Acid Solution, ibid. **97**:657, 1932.

15. Eagles, B. A., and Vars, H. M.: The Physiology of Ergothioneine, J. Biol. Chem. **80**:615, 1928. Baernstein, H. D.: The Determination of Methionine in Proteins, ibid. **97**:663, 1932.

16. Tillmans, J., and Philippi, K.: Ueber den Gehalt der wichtigsten Proteine der Nahrungsmittel an Kohlehydrat und über ein kolorimetrisches Verfahren zur quantitativen Bestimmung von stickstoff-freiem Zucker im Eiweiss, Biochem. Ztschr. **215**:36, 1929.

17. Dische, Z., and Popper, H.: Ueber eine neue kolorimetrische Mikrobestimmungsmethode der Kohlehydrate in Organen und Körpersäften, Biochem. Ztschr. **175**:371, 1926.

The next question arises as to whether or not the carbohydrates in each of the lenticular proteins are the same. By enzymatic action, an impure complex carbohydrate has been isolated from the whole lens protein, which had previously been freed from dextrose. So far, all attempts have failed to prepare a lenticular carbohydrate, which would give a precipitin reaction with serums from rabbits that had been made immune to lens extract.

The large molecular weights of the water-soluble proteins must be considered as playing a part in the colloidal behavior of the lens. The colloidal molecules of the proteins are retained by the walls of the lens fibers and by the capsule, while most of the smaller noncolloidal mole-

TABLE 7.—*Minimal Molecular Weights of Lens Proteins*

Lens Protein	Amino-Acid	Weight Containing One Molecule	Assumed Number of Molecules	Minimal Molecular Weight
Alpha crystallin	Arginine	1,548	16	24,758
	Histidine	4,247	6	25,482
	Lysine	7,807	3	23,421
	Tryptophan	24,020	1	24,020
	Tyrosine	3,409	7	23,863
	Average			24,300
Beta crystallin	Arginine	1,521	10	15,210
	Histidine	3,621	4	14,484
	Lysine	3,167	5	15,885
	Tryptophan	14,897	1	14,897
	Tyrosine	3,732	4	14,929
	Average			15,100
Albumin	Arginine	1,359	18	24,462
	Histidine	4,668	5	23,380
	Lysine	2,755	9	24,795
	Tryptophan	23,192	1	23,192
	Tyrosine	4,872	5	24,360
	Average			24,000

cules are diffusible. The increase of the permeability of the capsule in any manner, such as injury or swelling of the lens, tends to permit the water-soluble lens protein to diffuse at a rate inversely in proportion to the size of the protein molecule. It is interesting for this reason to attempt to find a probable molecular weight for these proteins.

The method of calculating the minimal molecular weights from analytic data is given in the following equation:

$$\text{Minimal molecular weight of protein} = \frac{\text{molecular weight of amino-acid} \times 100}{\text{percentage of amino-acid in protein}}$$

The minimal molecular weights estimated from the percentages of arginine, histidine, lysine, tryptophan and tyrosine are given in table 7. Unfortunately, on these proteins no physicochemical information such as equivalent and maximal combining capacities, osmotic pressures and permeability through standardized membranes is available for the estimation of the probable molecular from the minimal weight. The true

molecular weights of the proteins are probably integral multiples of their minimal molecular weights. If the molecular weights of alpha crystallin and beta crystallin are comparable to the weight of serum globulin and the molecular weight of lens albumin is comparable to that of serum albumin, the molecular weight of alpha crystallin may be considered as three times 24,300, or 73,000, beta crystallin as five times 15,100, or 76,000, and albumin as two times 24,000, or 48,000.

CONCLUSION

Quantitative analyses of beta crystallin, albumin and capsule were made. The four lens proteins and capsule possess a carbohydrate group in their structure. The minimal molecular weights of the proteins were estimated from the results of the chemical analyses.

The material was obtained through Mr. R. L. Fox. The technical procedures were performed with the aid of Mr. W. Tauber.

FIXATION AND VOLUNTARY NYSTAGMUS

A CLINICAL STUDY

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Fixation nystagmus, by which we mean nystagmus that results from fixing intently for some time on an object at rest, whether situated far or near, directly ahead or at an angle, is extremely rare. We have been unable to find reports of any such cases in either the English or the American literature. However, a few such cases have been mentioned by the Germans, and the subject was briefly discussed by Spiegel and Sommer,[1] Wilbrand and Saenger,[2] Brückner,[3] Oppenheim[4] and Fuchs.[5]

Voluntary nystagmus, on the contrary, while relatively rare, has been discussed for a long period of time, and to date there are over thirty cases which have found their way into the literature of the various countries. This condition is understood to be the production of a nystagmus by will power alone, that is, by concentrating on the idea and desiring the eyes to oscillate, with the resultant motion. This, to be of the true type, is independent of fixing the gaze in any direction or on any object.

During the past year we have observed two cases of nystagmus, one conforming in every respect to the fixation type as defined and the other to the pure form of what is defined in the literature as "voluntary nystagmus."

REPORT OF CASES

CASE 1.—J. C. S., a boy, aged 13 years, attending school, has been an inmate of an orphanage since babyhood. He has never seen either parent, and nothing is known of them or of his heredity. He has always been well physically and mentally,

1. Spiegel, E. A., and Sommer, I.: Ophthalmo- und Oto-Neurologie, Berlin, Julius Springer, 1931, p. 264.
2. Wilbrand, H., and Saenger, A.: Neurologie des Auges, Munich, J. F. Bergmann, 1921, vol. 8, p. 309.
3. Brückner, A.: Zur Kenntnis des sogenannten wilkürlichen Nystagmus, Ztschr. f. Augenh. **37**:184, 1917.
4. Oppenheim, Herman: Lehrbuch der Nervenkrankheiten, ed. 7, Berlin, S. Karger, 1923, p. 1929.
5. Fuchs, S.: Diseases of the Eye, Philadelphia, J. B. Lippincott Company, 1923, p. 341.

and has taken an active interest in sports at school. There is no encephalitic history, and no symptoms of a previous attack are present.

Examination showed the patient to be of average development for his age. His pupils were round and equal, and reacted promptly to light and in accommodation. The ocular movements, media and eyegrounds were normal. The visual fields for form and color were normal in both eyes. The vision was 20/20 in each eye; no refractive error was present, and the muscle balance was normal. The special senses were normal. The caloric response was normal in both ears. No stigmas of degeneration were found. The tendon reflexes of both the upper and the lower extremities were present, equal and normal in response. There was no sensory disturbance anywhere. Station and gait were normal. There were no evidences of any organic nervous disease. The blood pressure was 80 systolic and 60 diastolic. The heart, lungs and abdomen were normal.

Laboratory examination revealed normal urine, normal red and white blood cell counts, a normal hemoglobin content, a negative Wassermann reaction of the blood and a normal composition of the blood.

Mentally this boy has no vicious habits; he has a pleasing personality, sleeps well and is active, but is mentally retarded, owing in part to the oscillations of his eyes during the past three years on attempting to read.

About three years ago he observed that occasionally while studying his eyes, after fixing on an object, would suddenly "shake." This would be followed shortly by a painful closure of the eyes, and the motion would stop. Should he repeat his effort at reading, this phenomenon would be repeated after a short time. He tried to stop this in school, but it increased so that he was changed from close work to the manual training department, where his work does not require intense or long fixation on objects. This oscillation of the eyes can be brought on by fixing on an object in any direction, far or near. The oscillations are of a horizontal and rotary-horizontal mixture; they are rhythmic; the amplitude does not exceed 1 mm., and the frequency is between 400 and 500 per minute. The duration is but a few seconds, and the end is preceded by a rapid convergence of the eyes, associated with a painful closure of the lids and a shaking of the head. The eyes are then normal until he fixes on another object, when the process recurs. These oscillations can be produced not only when fixing on far or near objects with both eyes open, but when either eye is covered; also after a mydriatic or on employing prisms with bases set in any direction. The element of wish does not at any time play a factor in the production of the oscillations, and he cannot stop them except by convergence and closure of the eyes, both of which are automatic.

Case 2.—D. P., a woman, aged 21, single, a trained nurse, observed as a small child, when trying to develop some trick for the amusement of her playmates, that she could make her eyes "dance" at will. She recalls distinctly the onset, and states that she practiced doing this so that she could use it in parlor entertainment. To produce this condition, all that is required is to concentrate on the idea of making her eyes "jump," and shortly a nystagmus of rotary type, with some elements of a horizontal nature, will result. This nystagumus or oscillation can be started or stopped at will, and can be produced when one or both eyes are closed, the eyes moving as though they were open. This oscillation can be produced when looking straight ahead or when looking to either side, but it is easier when the patient looks toward the left. Prisms placed before the eyes with bases in any position have no effect on the oscillations. These oscillations are rhythmic and of short duration, and occur about three hundred times a minute. Should she "will" to continue the oscillations over many seconds, her eyes feel tired. However, after a few moments' rest, she can reproduce the condition.

The vision in each eye is 20/20. The muscle balance is normal. The media and fundi are normal. Fields for vision and color are normal. The pupils are round, equal and react to light and in accommodation. There is a slight hyperopia of + 0.37 sphere in each eye.

The family history is negative for any nervous or mental disease. The patient has five sisters and one brother, alive and well. Her father died from a postoperative complication. Her mother is alive and well. The patient had the usual diseases of childhood, and three years ago her appendix was removed. With the onset of maturity, she began having severe headaches of a migraine type. These occur two or three times a month and last from two to four days; they usually begin on the left side of the head, and terminate with severe nausea and vomiting. They are not associated with the menses, and have no influence on the ocular movements. There is no history of encephalitis, and no symptoms of a previous attack are found.

Physical examination revealed a well developed young woman, weighing 110 pounds (49.9 Kg.). The blood pressure was 110 systolic and 70 diastolic. The heart and lungs were normal. The special senses were normal. The tendon reflexes were all present and equal. There were no abnormal toe signs, no sensory disturbance of any type and no tremor of the fingers. Station and gait were normal. The caloric tests yielded normal results. Laboratory examination revealed nothing which deviated from the normal. The Wassermann reaction of the blood was negative.

The patient is sociable, a good mixer and diligent; she has a pleasing personality and has no tendency toward any eccentricities.

COMMENT

It will be immediately observed that the two cases differ in the mode of production of the nystagmus. The course is different and the phenomenon terminates differently. The fixation nystagmus comes on independently of any wish, but deep concentration apparently hastens the onset slightly. In the case of voluntary nystagmus the patient does not need to fix an object to produce the motion; in fact, she can produce the phenomenon by closing one or both eyes or by looking in any direction. The desire to have the eyes "dance" is sufficient, if she concentrates but a moment. Her case differs somewhat from many of the published cases in that respect. Several of those in the literature are what we should regard as a mixture of voluntary and fixation nystagmus. Most of the cases, however, come under the voluntary type. In Dodd's [6] case the patient fixed on an object and then wished for a moment, before the oscillations began. Friedenwald's [7] case began as an ocular nystagmus of the optokinetic type, and later the patient found that he could produce some motion of the eyes voluntarily by looking in any direction. Waddy's [8] patient was obliged to stare fixedly into the distance, and then by deep concentration and desire he could

6. Dodd, H. W.: Tr. Ophth. Soc. U. Kingdom **21**:244, 1901.
7. Friedenwald, Harry: Am. J. Ophth. **9**:364 (May) 1926.
8. Waddy, Granville: Ophthalmoscope **10**:316, 1912.

produce oscillations of the left eye alone. These were the only cases in which the element of any combination of fixation and a voluntary act were combined.

More or less pure cases of voluntary nystagmus have been described by Williams,[9] Lafon and Bonnet,[10] Coppez,[11] Pyle,[12] Benson,[13] Raehlmann,[14] Sterling,[15] Elliot,[16] Smith,[17] Lawson,[18] Gamble,[19] Brückner,[3] Wilbrand and Saenger[2] and others.

In any effort to explain these types of nystagmus one must of necessity consider the explanation of nystagmus in general. Many theories are postulated, but as yet no entirely satisfactory explanation of nystagmus is at hand. By nystagmus we understand a disturbance of the static condition of the eyes, in which the normal associated movements of the ocular globe become involuntarily rhythmic and present an associated tremor. Nystagmus in general is believed to originate from three sources: (*a*) ocular; (*b*) vestibular and (*c*) central—cerebellar or cerebral (Malcagni,[20] Spiller and Posey[21] and others).

In this communication we are especially concerned with the so-called ocular type, because we feel that our cases come under what various authors have agreed on as belonging to some disturbance of optic origin or of the neuromuscular mechanism of the eye. However, the various theories of nystagmus will first be considered.

The remarkable regularity and parallelism of the movements in nystagmus show that we have to deal with a perversion of the centers for parallel and parallel-rotary movements, and not with peripheral lesions of the muscles themselves or their nerves (Spiller and Posey[21]).

It is possible that nystagmus may be produced by alternating, just as normal fixation is produced by simultaneous, discharges of motor energy from the two sides of the brain. These centers are located in the cortex—the exact area is not yet isolated—and when impulses from both the right and left centers follow each other in rapid succession,

9. Williams, Henry W.: Ophth. Hosp. Rep. **5**:223, 1865.
10. Lafon and Bonnet: Rec. d'opht. **21**:320, 1909.
11. Coppez, H.: J. méd. de Bruxelles **13**:271, 1908.
12. Pyle, W. L.: Ophth. Rec. **19**:356, 1908.
13. Benson, A. H.: Roy. Lond. Ophth. Hosp. Rep. **10**:343, 1880-1882.
14. Raehlmann, E. V.: Arch. f. Ophth. **24**:237, 1878.
15. Sterling, A. W.: Ophthalmoscope **10**:71 (Feb.) 1912.
16. Elliot, R. H.: Ophthalmoscope **10**:70 (Feb.) 1912.
17. Smith, J. W.: J. Ophth., Laryng. & Otol. **14**:208, 1902.
18. Lawson, George: Roy. Lond. Ophth. Hosp. Rep. **10**:203, 1882.
19. Gamble, W. E.: Voluntary Lateral Nystagmus, J. A. M. A. **32**:483 (March 4) 1899.
20. Malcagni, A.: Ann. di ottal. e clin. ocul. **59**:1001, 1931.
21. Spiller and Posey: The Eye and Nervous System, Philadelphia, J. B. Lippincott Company, 1906, p. 273.

nystagmus results. Nystagmus may be interpreted as a type of imperfect or perverted fixation. In line with this theory, unilateral or dissociated types of nystagmus are those in which the reflex discharge from the associated centers which cause the oscillations is in some way allowed to affect one eye more than the other. Occasionally a muscular anomaly will explain the inequality of response. A number of authors (Fuchs,[5] Duane [22]) have expressed the principles of the foregoing theory in their writings.

Malcagni believes that whatever may be the nature of nystagmus, it is necessary to presuppose the existence of a rhythmic center which coordinates ocular movements, the changes of which result in the development of nystagmus. The stimuli which affect this rhythmic center and which activate it are those which control the motor apparatus of the globe. These arise from tonic association centers that regulate the associated movements of the eyes and preside over the function of binocular vision and of fusion of images in all directions of view. These associated centers receive their impressions in turn by various pathways (the vestibular, the auditive and the ocular), and may give rise to forms of vestibular, auditory or optical nystagmus. These impressions also are in relation with the cerebral cortex, which presides over the voluntary movements of the eyes. In these centers all the various stimuli are coordinated and subordinated.

Ohm [23] feels that there is not a great deal known about the cause of nystagmus. He feels that in mountain dweller's nystagmus and in voluntary nystagmus there are three possible causes, all interwoven: optical, vestibular and cerebral. Whether these arise from primary centers or from the cortex, he is not sure. He also demonstrated that concentration influences the character of the nystagmus in either case, showing the mental influence from the frontal lobe. He found that if the patient looks fixedly at an object situated at an angle, right or left, 10 cm. distant, there is an increase in the frequency of the nystagmus and a decrease in its amplitude.

Spiegel and Sommer [1] believe that fixation nystagmus may occur in normal people and also in people with degenerative stigmas. They said that it is best produced when looking at an angle, for then there is an imbalance in the ocular muscles in which the agonist has a stronger contraction than the antagonist, which results, in this type of person, in an oscillation or a nystagmus of the eyes of varying severity; it may occur in several directions, bilaterally or as a combination of these types with rotary nystagmus. They further believe that there is a similarity between muscle tremor of the skeletal muscles and the

22. Duane, Alexander: New York State J. Med. **5**:245, 1905.

23. Ohm, J.: Med. Klin. **21**:839, 1925.

oscillations of the eye. If a person with normal ocular muscle balance fixes intently for one or two minutes, the eyes usually oscillate; similarly, in fatigue of the body muscles, as after holding fast to an object for some minutes, a muscular tremor results. In the eyes this is called nystagmus. In both instances the result of repetition is that the tremor is more quickly produced, the amplitude is greater and the rhythm is faster. This condition can be easily produced with repetition in neurotic people. It has also been brought out in certain toxic states, and the more energy and the deeper the concentration on the process, the more frequently and easily is the condition produced. Spiegel and Sommer therefore regard the phenomenon purely on a functional basis, with fatigue as the precipitating factor. They feel that voluntary nystagmus can be produced by normal people, but not often; it usually occurs in persons with some stigmas. They regard it as a type of ocular nystagmus.

Lafon [24] regards voluntary nystagmus as a disturbance of the tonic equilibrium between the controlled muscles, owing to an excessive convergence. He feels that all voluntary movements of the eyes are associated ones, governed by the function of direction and convergence. These functions are of cortical origin, arising not from one center, but from several associated sensorial centers scattered through the cortex. They are coordinated by centers located probably in the mesocephalon, which transmits them to the oculomotor nucleus. There may exist, also, supranuclear, intermediary and subcortical centers.

Brückner,[3] after revewing all the cases of voluntary nystagmus up to 1917, and adding several of his own, agreed with our observation that all cases were purely voluntary. He was not certain that the term "voluntary" explains the response. It was his opinion that there is a strong innervation impulse sent to the muscles of both eyes, after which the eyes begin to oscillate. He did not think that it fully represents a voluntary act or response. He expressed the belief that only a few people can produce the phenomenon, and that such persons are susceptible to strong innervation and strong impulses affecting the eyes.

It would appear, then, that there are really two schools of thought relative to the mechanism of these types of nystagmus: One (Spiegel and Sommer,[1] Brückner [3] and others) considers that the nystagmus results wholly from physiologic responses to stimuli affecting the muscles of the eyes in neurotic or hysterical types of persons or in those held susceptible to strong impulses directed to the eyes with the idea of oscillation or motion in mind. Others think it a fatigue process, while still others liken it to a tremor of the skeletal muscles in fatigue.

24. Lafon, C.: Nystagmus, Ann. d'ocul. **153**:529, 1920.

The second school (Malcagni,[20] Ohm [23] and others) considers nystagmus a part and parcel of stimuli affecting the cerebral coordinating and rhythmic centers for the eyes, which are not definitely fixed centers, but are located in the cortex and subcortical centers in the mesocephalon.

COMMENT

It is evident to us that both our cases show what is regarded as the ocular type of nystagmus, though they present somewhat different manifestations of this rather unique condition. Ocular nystagmus manifests itself in many forms, which grouped together may be regarded as the result of some change in the eye itself, in some disturbance of its neuromuscular mechanism or in a faulty position of the eye, owing to faulty refraction or to some degenerative defect of the eye. The causative factors, thus, may be either physiologic or pathologic.

The nystagmus resulting from position (*endstellungs Nystagmus*), as from fixing the eyes in the limit of the field of vision, and optokinetic nystagmus, due to observing moving objects, as from a car window of a rapidly moving train, or observing high water-falls, are physiologic; while those types dependent on imperfect light plus bad position of the eyes, as in miner's nystagmus, mountain dweller's nystagmus and spasmus nutans, and those due to impaired vision of congenital origin or to disorders of acuity of vision or of vision itself, as in amblyopia, are regarded as pathologic.

Voluntary and fixation nystagmus and latent, unilateral and dissociated nystagmus are classed along with the ocular types, and have a more or less common factor in their production, but still the mechanism of the phenomenon in the various types is somewhat different.

In our case of voluntary nystagmus the condition appears to be the result of a sudden strong impulse applied to the muscles of both eyes with the idea of oscillation in mind; by the frequent repetition of these stimuli there has resulted the easy production of the movements at will. All this, however, presupposes a rhythmic center in the brain to coordinate the ocular movements. The stimuli, in the form of wish and concentration, affect this rhythmic center, in all probability in such a way that there is a change in the equilibrium, and alternating rather than simultaneous discharges of motor energy from the two sides of the brain occur with resulting nystagmus. It is barely possible, however, that fatigue may play a part in this case, as the patient usually turns the eyes somewhat to one side before the onset: in this way the imbalance of the agonist and antagonist muscles, due to fatigue, may permit the oscillations or tremor of the ocular muscles to ensue, with the resultant nystagmus. This belief is strengthened when we

bear in mind the completely negative findings in this case in the way of refractive errors, muscle balance and changes in the fields, and the absence of any demonstrable organic lesion of the eyes or brain.

In our case of fixation nystagmus the mechanism is somewhat different, as here there is no wish element, but merely the fact of fixation on an object at any point in the visual field. While early in this case it took longer to produce the nystagmus than at present, it would appear that there is a strong element of fatigue, the muscles for maintaining the static condition of the globe becoming unequally fatigued and an oscillation resulting. This rapidly increases until convergence occurs, which becomes painful; this is followed by a sudden closure of the lids, and the oscillations suddenly cease, the eyes immediately resuming their normal position. This explanation can be considered analogous to the muscle tremor theory of Spiegel and Sommer, and the condition may thus be more the result of a neuromuscular mechanism than the result of a strong mental stimulus affecting the rhythmic center of the cortex in such a way as to give rapid alternating, rather than simultaneous, discharges from this center. Again in this case we have the complete absence of all organic ocular and neurologic signs and a perfectly normal muscle balance of the eyes, with no refractive errors and no disturbance of vision whatever.

The final analysis thus would indicate that in both of these cases the nystagmus is functional, rather than organic in origin, but that it is produced by somewhat different mechanisms: in one case rather by strong desire, with concentration; in the other by fixation on an object before the oscillations begin. The one nystagmus terminates at will; the other, only after rapid convergence and closure of the lids. The duration of each is from twenty to thirty seconds, the amplitude and rapidity of the oscillations are about equal in each and both patients feel fatigue about the eyes after the phenomenon has passed.

SUMMARY

1. Two cases of nystagmus are herein described, one of the so-called fixation type and the other of the voluntary type.

2. The patient with fixation nystagmus produces it by fixing on an object in any point of the visual field; soon a rapid oscillation occurs, which is followed by sharp convergence and pain and then by a sudden closure of the lids, with immediate return of the eyes to normal.

3. The case of voluntary nystagmus is that of a young woman, who, by concentrating on the wish to oscillate her eyes or to make them "dance," with or without the eyes closed or when looking in any direction, shows an oscillation of both eyes. This can be stopped at will, but if it is unchecked the eyes are fatigued in about thirty seconds and the process stops.

4. We feel that there is probably the same underlying principle in the production of both types of nystagmus. The sending of strong stimuli from the rhythmic center of the cortex, producing alternating rather than simultaneous discharges of energy, is one explanation.

5. The process may also be explained as the result of fatigue of the muscles resulting from the primary fixation of gaze plus frequent repetition of the process, with resulting imbalance between the agonist and antagonist muscles and oscillations.

6. In either event, we feel that both types belong to what is known as ocular nystagmus, and that both are functional in origin.

ABSTRACT OF DISCUSSION

Dr. John H. Dunnington, New York: Duane considered nystagmus due to a perversion of the centers for parallel and parallel rotatory movements, and referred to it as a kind of perverted and imperfect fixation. If nystagmus is due to a perversion of fixation, it is reasonable to expect that in some cases the oscillations will become worse on attempting fixation. Macular lesions and central corneal or lenticular opacities are frequently encountered in this type of nystagmus. All of these conditions render fixation more difficult.

Voluntary nystagmus is unique, and its occurrence has been frequently noted in the literature. Bruckner, an oculist, who possessed this power, described the sensations he experienced during the oscillations as those of marked innervation of convergence and of accommodation, and he compared them to those felt when the arm is held in strong flexion. Bruckner, Weekers and others have referred to contraction of the pupil during the oscillations. These observations would lead one to think that the nystagmus resulted from the impairment of vision produced by the transitory spasm of accommodation, yet Smith has recorded a case of voluntary nystagmus in which the oscillations could be produced while the eyes were closed. Furthermore, the unilateral cases reported by Pyle and Ball are not easily explained by the visual theory. With such conflicting evidence, it seems most plausible to assume that Dr. Luhr is correct in his contention that there is a center controlling the rhythmic movements of the eyes, and that voluntary nystagmus results from some functional disturbance of this center.

Dr. E. E. Blaauw, Buffalo: The old idea of Alt, that because the macular region is involved the patient wants to shift his eyes to see better, is strongly contradicted by Ohm. He directs attention to the cases of monocular nystagmus which show, monolaterally—when the eye is in motion—a distinct diminution of vision, which improves as soon as the eyes are used together and thus lose the nystagmus.

Dr. Edward Jackson, Denver: In fixation nystagmus, the voluntary effort is that of fixation. In the nystagmus that might be called involuntary nystagmus, a certain psychologic element enters; the innervation has probably been acquired by practice. It seems to me that this is an experiment that should be connected with nystagmus. I have felt that poor vision is pretty closely connected with nystagmus, and is not merely an effect. I have seen nystagmus in cases in which there was no poor vision, with an error of refraction; I have also seen a few cases in which the nystagmus gradually cleared up after correction of the error of refraction, and some in which the nystagmus decidedly diminished and remained so for several years.

Some one remarked that nystagmus occurs more frequently than is suspected. Examining eyes for nystagmus with the ophthalmoscope, fixing on the back of the eye and trying to keep the eye fixed, in a fair number of cases, if kept up for a short time, will cause a slight nystagmus. With the ophthalmoscope it is possible to detect nystagmus that is very hard to find by any other method of examination, and it is possible to measure by the movements of the vessels and the movements of the optic nerve the amount of nystagmus that occurs.

Clinical Notes

ANALYTIC DUCTION TESTS

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The classic conception of duction tests is that they determine the amount of prism base out, in, up or down which the eyes can overcome in order to maintain single binocular vision. They are for this reason also referred to as "breadth of fusion tests." This conception is purely quantitative and ignores several qualitative elements involved in an analysis of the meaning of duction tests and findings.

There are three qualitative phases in a duction test, which can be best exemplified by an illustration. Suppose one makes a near-adduction test, using a tiny spot light at 35 cm. as a target. To begin with, one must make sure that the patient has single binocular vision, and that he sees the light single with both eyes. One then inserts prisms base out of increasing power or uses rotary prisms until the light doubles up. Usually the patient sees the prism break into two. For this reason it is appropriate to call the point at which this doubling occurs the break point. If it takes place at 20 prism diopters, the record is "near adduction-break at 20 prism diopters." A slightly stronger prism separates the images considerably. In making the test, it is advisable to increase the prisms somewhat beyond the break. By reduction of the prism power, the two images approach and finally merge again into one. This point is appropriately called the recovery point. If it took place at 16 prism diopters, the record would be "near adduction, break at 20, recovery at 16 prism diopters," or simply "20-16 prism diopters."

It is now apparent that there are at least two values in every duction test: Adduction and abduction for distance or near will show one prism value at the break point and a different prism value at the recovery point. A duction entry on the record card must include both values in some such manner as "near-adduction 20-16," the figures of the foregoing illustration being used.

What is the significance of each of these values? In the illustration the break point represents the maximum convergence effort which can be made in order to maintain single vision. It has a purely quantitative significance; it is a limiting value, to use a mathematical term. The recovery point obtained after considerable separation of the images and a moment's rest shows the maximum convergence effort which can be *sustained* over a period. It has a qualitative significance. The gap between the break and the recovery points represents the interval between sustaining power and exhaustion. For measurement of reserve power the position of the recovery point is of greater significance than that of the break point. A high recovery point, for example, in the near-adduction indicates good reserve sustaining power of the convergence.

A low recovery point indicates qualitatively a low reserve. Thus a near-adduction of 20-16 is more likely to overcome without strain a near-exophoria than a near-adduction of 25-10 prism diopters. The same applies to the other ductions.

There is one other point to be considered in making qualitative duction tests. Suppose that instead of having used a point of light as the target one had used a very small, sharply outlined cross or letter. On careful testing one would find that some time before the letter doubled it became blurred. For example, the letter may have been single and clear up to 12 prism diopters; at 13 it blurred; at 20 it doubled, and at 16 it recovered. There are now three values which must be recorded. There is, in addition to the break point and recovery point, the blur point, that is, the stage at which the target though seen single is seen blurred. The entry would be, "near-adduction, blur at 13, break at 20, recovery at 16 prism diopters," or briefly, "13-20-16 prism diopters." The recovery and blur points always lie below the break point. But with reference to each other the blur or recovery point may be higher.

Duction findings with regard to each of these three points of reference are far more informative than the simple test in which only the break point is recorded. It has been shown that the break point represents the momentary effort and has mainly a quantitative significance, and that the recovery point represents the maximum sustained effort and has mainly a qualitative significance. What is the significance of the blur point?

Briefly, the position of the blur point indicates the extent of "hook-up" between the accommodation and the convergence, for at the moment when the target begins to blur, the accommodative function is coming into play, with increase of activity in the near-adduction test and decrease of activity in the near-abduction test. If, for example, in the near-adduction the blur and break points are very close, say, 17 and 20 prism diopters, it indicates, for one thing, a loose hook-up between the two functions. The accommodation came into activity only at the height of the increased convergence; up to 17 prism diopters the convergence was free and increased without involving accommodative activity. This free convergence, it may be added, consisted of the fusional convergence and the proximate convergence; i. e., the convergence produced through the fusion faculty and the convergence produced through the consciousness of nearness. On the other hand, if for example in the near-adduction the blur and break points are far apart, say, blur at 6 and break at 20 prism diopters, this indicates, for one thing, a tight hook-up between the accommodation and the convergence. Here the loose convergence was only 6 prism diopters; from that amount to the breaking point it was accompanied by accommodative activity.

The extent of free and forced play between the accommodation and the convergence is an important element in the determination of corrections for lenses for ametropia, presbyopia, subnormal accommodation, muscular imbalances and similar conditions. Its importance has long been recognized. In the works of Donders, Landolt and the other earlier masters stress is placed on determination of the extent of positive and negative relative accommodation and relative convergence. The analysis of duction tests, especially at near, from the standpoint of the blur, break and recovery points is a further extension of this work.

Generally speaking, the eyes seem best balanced when the blur point is about half the break point. For instance, when the break is at 20 prism diopters, the blur point should lie around 10 prism diopters, but reliable norms for these points are still to be formulated. There is, however, this to be said with reference to the position of the blur point. While the break and recovery points can be determined for almost every patient, the determination of the blur point is more difficult. It requires active cooperation, great care and a more or less intelligent patient if reliable results are to be secured. The distorting effect of the prisms is also a disturbing factor. It is difficult if not impossible to separate the blurring due to the distortion of the prisms from the blurring due to a change in accommodation. The effects of the prism distortion may be somewhat lessened by the placing of the bulk of the prism power before one eye, preferably the nondominant eye. The blurring due to the prisms will not be so marked as long as the eyes are properly accommodated for the distance, and the dominant eye receives a clear image.

There is, of course, another phase in the interrelation of accommodation and convergence, namely, the relative accommodation, positive and negative. In tests for this with minus and plus lenses, respectively, one likewise stops at the point of blurring. There is thus a blur point in the tests for relative accommodation as well as in the tests for relative convergence included in the full duction tests. In the tests for relative accommodation the blur point is at the limits of the relative convergence. In the duction tests it is at the limits of the relative accommodation. But the cause of the blur is different in the two. In the relative accommodation tests, the blur results from a *non-change* in accommodation concomitant with a *non-change* in convergence. In the duction tests, the blur, aside from the effects of prism distortion, results from a *change* in accommodation concomitant with a *change* in convergence. The two values are, however, closely interrelated.

It is apparent that the extent of accommodative activity corresponding to changes in convergence can be altered by way of stimulation or repression, by means of minus or plus lenses. These will generally tend to shift the position of the various points, blur, break and recovery, and may be used to bring about a more comfortable hook-up between the accommodation and convergence. The time and effort spent in making these tests will often be well repaid in the clearing up of an obscure case.

SUMMARY AND CONCLUSION

In making duction tests one must take into consideration that there are generally three elements involved, and that each of these has a special significance. There is the blur point, the stage at which the accommodation comes into play with a change of convergence. The postion of this blur point relative to the maximum at the break point indicates the kind of hook-up between the accommodation and the convergence. Then there is the break point, the stage at which an absolute maximum of convergence or divergence is produced, indicating a quantitative limiting value to the activity involved. Finally, there is the recovery point, the stage at which single vision is again resumed. This value shows the sustaining power of the function involved and is largely of qualitative significance. Duction tests made with a view to these three elements are helpful in every case and are especially useful in clarifying obscure cases of refractive and muscular anomalies.

TREATMENT OF AFTER-CATARACT

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This is a perennial subject. How numerous are the different ways of needling, yet they are all essentially the same, each claiming advantages and each claiming to be the last word on the subject until another so-called new one comes out. This shows, as has long been evident to me, that no one of them is really satisfactory. One of my early victories was in a patient (with albuminuria, aged 60) who came to me with a dense after-cataract, the iris being tied down to it all around. The cataract had been extracted by one of the most competent operators in the world by the capsulotomy method, and he told the patient that nothing more could be done. Wisely he had not attempted to needle it, for in such an after-cataract, needling means death to the eye. I atropinized the patient heavily, made an incision as if for an iridectomy, caught a piece of iris and excised it. I then inserted an iris repositor through the opening into the posterior chamber and separated the attachment to the after-cataract all around. In these cases there is always a posterior chamber. The pupil, when released, dilated, and I inserted iris forceps and took out the after-cataract. There was no escape of vitreous, and all went as well as the most ambitious could desire. A week later I sent the patient home with full normal vision with spherical lenses. I gave him the after-cataract in formaldehyde, and advised him to ask the original operator to refract him for spectacles a few weeks later, and to show him the after-cataract if he desired to see it. Since that day I have advocated extraction of after-cataracts instead of needling (see my monograph on cataract,[1] among other papers).

I was interested a short time ago when looking over Indian Civil Hospital Annual Reports (in the Record Room of the India Office in London) for the Indian Empire, covering a number of recent years.[2] These reports are extensive and in great detail, including all hospitals except a few missionary ones. Extraction of an after-cataract is almost universal now in India; there is not a case of needling recorded except in one hospital It may be of interest to some readers to think over the matter.

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1. Smith, Henry: Statistics of Cataract Extraction, Arch. Ophth. **8**:595 (Oct.) 1932.

2. There is not such a mass of systematized material in any other country in the world.

Ophthalmologic Review

STATUS OF CARDIOVASCULAR RENAL DISEASES

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This summary attempts to present the internist's attitude toward the problems of cardiac, vascular and renal disease. Little, if any, of the matter contained is original, and some of it is controversial. The disease pictures described do not differ from one decade to another, but the methods of approach, the interpretation of signs and symptoms and the technic of treatment show variations, possibly cyclic.

TYPES OF CARDIAC DISEASE

Little has recently been added to the knowledge of congenital disease of the heart. The existence of the disease still presages a short life span; 60 per cent of patients with congenital cardiac lesions die before their fifth year, and only 8 per cent pass the age of 30. Of those who live, the majority have minor septal defects or stenosis of the pulmonary artery, with some compensatory venous arterial communication, such as a patent ductus arteriosus. Respiratory infections are responsible for many deaths, and bacterial endocarditis is common on damaged valves.

Rheumatic disease of the heart, as now interpreted, may be the outcome of classic rheumatic fever, chorea or less well defined evidences of infection, such as repeated tonsillitis. At one or another stage, the entire structure of the heart is involved: endocardium and valves, muscle and pericardium. In the most severe forms, a true pancarditis is found. The valvular lesions are, in part, at least, the result of healing of the endocarditic process, and result usually in stenosis of the mitral valve or incompetence of the aortic valve. These lesions may not be demonstrable until months after the close of an acute attack of arthritis or chorea. The familial incidence of rheumatic fever, and hence of rheumatic disease of the heart, has become more apparent, as has the geographic distribution of the illness; it is a disease of northern cities, and is less and less common as one approaches the tropics.

Syphilitic disease of the heart, which is indistinguishable from syphilitic aortitis, presents as a rule between the ages of 35 to 50, from

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ten to fifteen years after acquisition of the disease. It shares with syphilis of the central nervous system first place as an end-result of syphilitic infection. Original lesions in the heart itself are dwarfed in importance by those in the aorta: aneurysms or aortic insufficiency. It seems more common in persons who work with their hands, and is far more common in men than in women.

The "thyroid heart" represents a relatively new conception. Cardiac involvement in severe toxic thyroid disease has long been known, but it is only recently that adequate attention has been given to the cardiac disease associated with lesser toxic adenomatous (or better, nodular) goiters. Presenting chiefly in women, these cardiac lesions are more common in the fifth decade, and may be associated with evidence of mitral stenosis of rheumatic origin or of hypertensive disease. Their most important feature is that removal of even mildly toxic thyroid nodules is often followed by startling improvement and even apparent cure.

"Chronic myocarditis" is a misleading term, since true muscular inflammatory changes are uncommon. More accurate is the designation hypertensive or arteriosclerotic conditions of the heart, though one can only partially separate these degenerative diseases into the two groups. In the first, arteriosclerosis of the aorta and the coronary arteries dominates the picture. Angina pectoris and the dramatic syndrome following occlusion of a major coronary branch are common, and high blood pressure is not necessarily found. Rare in the late thirties, this picture is more common between 45 and 60 years of age, and is rather more common in men. The patient with hypertension almost always carries a component of coronary sclerosis and is subject to the various phenomena associated with atheroma. However, he (or better, she, for this form of heart disease is more common in women) may show a picture of gradual heart failure, with remissions and exacerbations which almost approximate those of mitral stenosis. I may say here, and shall repeat later, that hypertensive disease of the heart is the most common end-result of the disease, hypertension.

TYPES OF VASCULAR DISEASE

Apart from syphilis of the aorta, vascular syphilis is largely confined to arterial disease of the central nervous system and is often associated with gummatous meningitis. While dementia paralytica or tabes may be associated, vascular and meningeal disease may exist without concomitant deeper nerve cell changes. Syphilis of the arteries elsewhere is a rarity.

Atheromatous or arteriosclerotic vascular disease affects cerebral vessels next in frequency to the aorta and coronary arteries. Among

visceral structures, the pancreas seems relatively susceptible, and diabetes in later life is more common in arteriosclerotic persons and families.

Hypertension as a disease can be divided into benign or essential hypertension, or hyperpiesis, and malignant hypertension. The latter is an uncommon disease which is relatively more frequent in younger persons than the benign form, appearing even in childhood. Necrotic changes in arterioles characterize the pathologic picture, and a short, stormy course with renal death is usual. The common form of hypertension appears at the climacteric in women, and a little later in men. One speaks roughly of the degree of elevation of the systolic pressure as "low level" when under 180 mm. of mercury, as "mid-level" between 180 and 220 mm. of mercury and as "high level" when above this. These general points may be emphasized: First, the disease is more common in women than in men; second, the earlier the age of onset, the more serious is the course; third, cardiac disease is the most common termination, and fourth, high pressure levels are not incompatible with a normal life span. While renal lesions and their results often play a part in the final picture, only 15 per cent of persons with hypertension die of nephritis, as against 25 per cent with apoplexy and 60 per cent with cardiac disease. The implications as to treatment will develop.

Secondary to renal disease, usually hemorrhagic nephritis, there may be both arteriosclerosis and hypertension. The associated renal picture naturally modifies, according to its severity, the general course of the disease.

TYPES OF RENAL DISEASE

Congenital cystic disease of the kidney may lie latent for years and eventually exhibit itself only when renal insufficiency has advanced to a stage of early uremia. As in other types of renal insufficiency, secondary vascular disease with hypertension may develop. Renal damage from obstruction (stones, prostatic hypertrophy, etc.) is often much less in extent than it appears to be before relief of the block. Marked recovery of renal function is the rule after successful prostatectomy, and it must be assumed that no great loss of glomeruli has taken place. Associated or independent infection enormously reduces the available renal tissue. Nevertheless, frank renal failure is an uncommon event in these patients; they usually succumb to generalization of the infection. Of all this group, prostatic obstruction is of chief importance, and its recognition as a factor in cardiovascular-renal pictures is of the utmost importance.

The work of Addis, Volhard and Van Slyke is reflected in the modern classification of Bright's disease. Hemorrhagic nephritis is usually associated at onset with streptococcic infections, runs a long course with exacerbations, remissions and latent stages and is charac-

terized at the onset by frankly bloody urine and throughout its course by a tendency to excrete an increased number of red cells. While at the onset and late in the disease edema may develop, the usual end-picture is that of nonedematous nephritis, with progressive destruction of glomeruli and progressive renal insufficiency, as measured by output of dye, nitrogen retention and Van Slyke's urea clearance method of estimating surviving glomeruli. The Addis count measures more accurately than any other test the status of renal inflammation. Degenerative nephritis includes the sequelae of mercurial poisoning, lipoid nephrosis and amyloid nephrosis. These diseases have in common marked albuminuria, excretion of casts, without hematuria, and a notable tendency to edema. Hypertension is rare, and the uremic state a complication. Amyloid kidney is a part of generalized amyloidosis, as a rule, most commonly in the course of chronic infections, such as tuberculosis. The lipoid infiltrations of the kidney are more common in childhood and have been associated with persistent focal infections, especially those due to the staphylococcus. Changes in plasma proteins are the rule: depletion of albumin through abnormal excretion plays the chief rôle in the production of edema. All the renal complications of pregnancy pathologically belong in the degenerative group, though clinically they may be characterized only by hypertension and the convulsive state. When renal dysfunction persists after pregnancy, it is usually due to underlying hypertensive disease or to hemorrhagic nephritis, aggravated by the gravid state. The arteriosclerotic nephritis group includes the end-results of benign or essential hypertension in those instances in which the kidney is chiefly affected and the rapidly developing nephropathy termed malignant hypertension, which is pathologically an acute necrotizing arteriolitis. In both diseases renal function is impaired by gradual vascular occlusions, developing slowly or rapidly, depending on the type. Edema and hematuria are uncommon. This classification seems likely to replace the older division into acute, subacute and chronic nephritis, since it represents a closer approach to an etiologic division and permits a closer estimate of the pathologic findings. With few exceptions, acute and subacute nephritis are of the hemorrhagic type. Since edema may develop in hemorrhagic or arteriosclerotic nephritis as well as, commonly, in the degenerative types, the differentiation of chronic nephritis into classes with and without edema seems inadequate. "Vascular hypertension progressing into nephritis" (Christian) is synonymous with arteriosclerotic nephritis.

METHODS OF STUDY IN USE

The methods of study in use include: physical examination, hemoglobin determination, roentgenologic estimation of the size of the heart,

simple tests of the urine, electrocardiographic examination, phenolsulphonphthalein test, Addis count, determination of the nonprotein nitrogen of the blood and the plasma proteins, and urea clearance test.

There is no substitute for a careful physical examination. The majority of the tests listed, or of other tests in medicine, merely amplify the observations made at the bedside or in the consulting room. One notes edema, dyspnea, the heart rate, the color of the skin and mucosae, the state of the retinal and peripheral vessels, the condition of the teeth and tonsils, and sometimes of the sinuses; one examines for evidence of congestion in the lungs and liver; one notes the force of the apex beat of the heart, its percussion measurements, its sounds and the regularity of the rhythm. Allowing for psychic elevations, one determines the brachial blood pressure. Finally, in men, one notes the amount of prostatic enlargement by rectal examination. The hemoglobin determination and roentgen examination confirm the clinical estimates already made of the color and size of the heart. Of the simpler tests, those of the acidity and specific gravity of urine are important; the patient with severe glomerular damage cannot usually secrete an acid urine, nor maintain a normal specific gravity. If the morning urine, after a "dry" diet for afternoon and night, reaches a concentration of 1.020, renal damage cannot be extreme. The urinary sediment reveals casts and red blood cells only in acute or subacute hemorrhagic nephritis. The Addis count is a more delicate estimate of these formed elements. For convenience, the output of urine is made small by reducing the intake of fluids for an entire twenty-four hour period; in order to insure an acid urine (for preservation of casts), no fruit or leafy vegetables are allowed for the same period. Collection of urine is then made for a twelve hour period, usually overnight, the catheter being used in women to avoid obvious contamination. The urine is measured, and 10 cc. is centrifugated, with subsequent dilution of the sediment to 1 cc.; the casts and the red and white blood cells are counted in an ordinary hemocytometer chamber. In the normal person, up to 400,000 red blood cells and 4,000 casts may be excreted in twelve hours. Cases of hemorrhagic nephritis can be followed especially accurately by this test; one can make certain deductions from the type of cast found as to the type of nephritis. While involving no difficult principles, the test is accurate only after practice, and one should perform several on oneself before venturing to record results on patients. The phenolsulphonphthalein test is a measure of glomerular excretion of a nontoxic dye. One cubic centimeter of the standardized solution is injected intravenously, and the output for one-half hour periods and for a total of two hours is measured in a colorimeter against solutions of known concentration. This test is essentially crude, and has value chiefly in surgical problems of the kidney. The

nonprotein nitrogen of the blood represents retention of material ordinarily excreted, and is also a measure of glomerular activity. The urea clearance test (Van Slyke) is an estimate of the number of cubic centimeters of blood cleared of their urea per minute by the kidneys, and is the most accurate test of glomerular function known. The blood urea and the amount of urea excreted in standard time (and volume) in the urine are determined, and comparison is made with an expected normal excretion of the urea content of 75 cc. of blood. In arteriosclerotic nephritides, the blood flow through the renal parenchyma undoubtedly enters into the factor of excretion. This delicate test shows impairment in persons with hypertension who have normal renal function by all other tests, and hence is to be interpreted with some reserve until more accurate correlation can be established between altered values and eventual prognosis. Determination of the plasma proteins, albumin and globulin has interest and value chiefly in relation to edematous patients. Their deficit is ordinarily in albumin; when the albumin falls from a normal value of 5 per cent to about 2.5 per cent, the osmotic pressure of plasma is inadequate to keep fluid within vessel walls, and edema results. The globulin is fairly stationary at about 2.5 per cent. It is the loss of substances which exert osmotic pressure and not merely decreased albumin which makes for renal edema. One should state here that edema may depend on cardiac failure with increased venous pressure, or on the complicated factors which produce acute Bright's disease. It is chiefly in the study of chronic nephritis with edema, or of nutritional edema, that the test is of value. The electrocardiogram is used now more for study of the form of the heart's action current than for analysis of its rhythm. One can follow damage to the heart muscle in acute rheumatic fever or estimate the amount of damage done in the patient with arteriosclerosis or hypertension, and (though this is not often of practical benefit) localize the muscular infarct in patients who have had a major coronary occlusion. It is easy to be too liberal in interpreting electrocardiograms; it is well to remember that with any new technic one is prone to find many abnormalities which time teaches one occur in normal persons.

The amount of study indicated in a hypertensive patient may pertinently be discussed at this point. One wants to know especially: (1) How poor is renal function? (2) How poor is the heart muscle? The history, the physical examination and simple examinations of blood and urine usually give clues. Routine electrocardiograms and urea clearance tests are impractical in office practice. However, they should be done in patients who are in hospitals. In office patients, a capacity for variation in urinary specific gravity, nonprotein nitrogen and hemoglobin should be almost a routine observation. When one gets a "lead" toward

increasing cardiac disability, the roentgen silhouette of the heart is often helpful, and the electrocardiogram may give evidence of more impending than present failure. In patients with anemia, marked retinitis and alkaline urine of low specific gravity a low phenolsulphonphthalein excretion may confirm one's estimate of poor renal reserve. In general, unless there is definite evidence of prostatic obstruction, surgical consultations regarding teeth, tonsils, sinuses, etc., are wasted. If, however, there is evidence of hemorrhagic nephritis (blood in the urine, even microscopic evidence, or a high Addis count), focal infection should be investigated. Removal of foci will not affect the blood pressure, but may remove one factor tending to increase the renal disability. One additional comment on the simplest laboratory test performed, examination of the urine for albumin: In acute hemorrhagic nephritis large amounts of protein are present and also in the degenerative types of nephritis. However, in the most severe renal insufficiencies of terminal hemorrhagic or arteriosclerotic renal disease, there may be scant or almost no albumin excreted. Cardiac failure and chronic passive congestion are responsible for albuminuria in many patients, and appropriate treatment of the cardiac factor may clear the urine. Especially in the person with hypertension, the presence of much albumin, unassociated with casts or red blood cells, should call to mind a cardiac complication.

PROGNOSIS

Prognosis is really a guess at an answer to two questions: How long will the patient with this disease survive, and how is he likely to die? It has already been stated that persons with congenital cardiac disease rarely survive their tenth year. Infections (pneumonia, bacterial endocarditis) rather than heart failure cause most of the deaths. In rheumatic disease of the heart sudden death may occur from pulmonary edema in mitral stenosis, or without any reasonable explanation in aortic stenosis. The patients with mitral stenosis, however, more commonly die after progressively severe and intermittent bouts of circulatory failure, with symptoms usually developing before 30 years of age. Rheumatic aortic insufficiency is often unknown to the patient until the late thirties or forties; when symptoms develop, the course is rapidly downhill. Syphilitic aortitis presents symptoms in the forties (about fifteen years after infection), and the course is often rapid. Sudden unexplained death is a feature of this disease. In the patient with an arteriosclerotic heart, major coronary occlusion is likely to occur with or without warning, and he may succumb in such attacks, often with epigastric pain, which earned the soubriquet "acute fatal indigestion" from older physicians. While the prognosis ad vitam in a patient with a recognized nonfatal cardiac infarct is poor, and heart failure is frequent after such

events, many patients survive for years, in comfort, and even may lose anginal pain, probably in consequence of a decrease in blood pressure. The thyrotoxic heart fails gradually, and gives ample time for recognition and adequate treatment.

In hypertension, the younger the patient, the more likely does the disease prove severe and rapidly progressive, and the more likely is renal death. In so-called malignant hypertension, one or two years is the average life expectancy. In older patients with hypertension, cardiac deaths—sudden, from coronary accidents, or gradually, from heart failure—predominate. Apoplexy comes without warning at any time in the course of arteriosclerotic or hypertensive vascular disease; there may be no prodromal symptoms, and there are no adequate preventive measures.

The end-results of nephritis depend on the type of disease, the age and the duration. Many children or young people with hemorrhagic nephritis are entirely cured, holding good glomerular function; others maintain inflammatory changes for years and gradually develop renal insufficiency and uremia, ending in death. Here the Addis count aids enormously in prognosis. An acute exacerbation of hemorrhagic nephritis, however, may rapidly change a relatively quiescent to a fulminating, fatal disease picture. Hence active, chronic active and chronic inactive stages are encountered. The natural end-result of arteriosclerotic nephritis is uremia, and as has been said, the younger the patient the shorter is the course. Patients with degenerative nephritis seem unusually susceptible to infections, and in childhood the condition is often caused by pneumococci. Full recovery is rare.

GENERAL PRINCIPLES OF TREATMENT

Cardiac Disease.—So far as preventive treatment is concerned, one can only reiterate the truism that it is the infections only which are preventable. Since it is often the repetition of attacks of rheumatic fever which finally produce cardiac disease, all measures toward the prevention of such attacks are worth while. Children in susceptible families should probably undergo tonsillectomy early; minor colds should be respected and perhaps, when possible, these persons should live in uniformly warm climates. Once the disease is established, tonsillectomy has doubtful value. It is too soon to tell whether vaccine therapy in the acute phases of the disease is worth while. I am not sufficiently sure of the etiologic relation of streptococci to the disease to be enthusiastic, on theoretical grounds alone, about vaccine therapy; it is too soon to tell empirically. It seems fairly certain that salicylates control symptoms only and do not affect the ultimate outcome. The prevention of syphilitic disease of the heart is a problem in the prevention of syphilis and in early, thorough and intelligent treatment.

It is well established that rest forms the basis of treatment in all established cardiac disease. This means that the work incidental to play must be restricted, as well as the effort necessitated by work. The dietary treatment of cardiac disease involves, first, loss of weight in overweight persons; second, restriction not only of salt but of all unnecessary sodium compounds (for it is the sodium and not the chloride ion which binds fluid); third, restriction of excessive intake of fluid, and fourth, in phases of decompensation, the administration of adequate carbohydrate in readily assimilable form. It might be added that multiple small feedings are preferable to large meals, especially in arteriosclerosis (gastric distention may precipitate coronary attacks), and that small quantities of alcohol seem at times to have a favorable effect on hypertensive and arteriosclerotic diseases of the heart. Digitalis is the foundation of drug therapy. It is used not only in auricular fibrillation, but in all patients with regular or irregular cardiac rhythms who have evidence of right-sided heart failure (venous distention, congestion in the lungs, liver or peritoneal cavity, edema, etc.). To augment the loss of cardiac anasarca, the diuretics in favor are theophylline, mersalyl (a mercurial, which must be given parenterally) and certain inorganic salts, ammonium chloride and potassium citrate especially, which seem to have the capacity of displacing retained sodium ions from protein aggregations. For acutely developing, right-sided heart failure, especially with high venous pressure, bleeding is returning to favor. The xanthine derivatives have been found to have distinct value in the treatment of coronary sclerosis, in either the frank arteriosclerotic or the hypertensive heart. Theophylline (and other preparations) will experimentally increase blood flow through the coronary arteries, and clinically relieve some sufferers from angina or from dyspnea associated with left-sided cardiac failure.

Of great importance is the recognition of the "thyroid heart," or of hyperthyroidism associated with other forms (rheumatic, hypertensive) of heart disease. In any patient with heart disease and a goiter, the burden of proof is theirs who oppose operative removal of the struma. The results obtained may approach the miraculous. It should be emphasized that metabolism tests are notoriously unreliable in cardiac disease, and that conclusions drawn from slightly elevated basal metabolic rates are doubtful in value.

As the knowledge of cardiac disease increases, the treatment of irregular heart action has changed. Auricular flutter and ventricular tachycardias are treated with digitalis and quinidine, respectively, but there is much less concern over the problem of stopping the more common arrhythmia, auricular fibrillation. There are conditions under which it may be a favorable rhythm.

Vascular Disease.—The treatment of established syphilitic arteritis is discouraging in prospect and usually in result. Nevertheless, in early cases of meningovascular disease, in aortic pain and, with reservations, in aortitis generally, it seems worth while. The older drugs, iodides, mercury and arsphenamine, have been augmented by another heavy metal, bismuth, and an arsenical capable of being administered intramuscularly, bismarsen. Under suitable treatment, patients with meningovascular syphilis can be much benefited. In many cases of aortitis, pain can be relieved, and there is evidence that the prognosis of syphilitic disease of the heart in general is somewhat improved under treatment. The problems of arteriosclerosis, apart from arteriosclerotic disease of the heart, are still unsolved. The attack on benign hypertension has been sharper. First of all, it has become apparent that the effect of most drugs in reducing blood pressure is due either to faulty observation or to psychologic factors alone. This much can be affirmed: No drug yet announced will uniformly and permanently reduce high blood pressure. Neither iodides, sulphocynates, nitrates, nitrites nor plant nor organ extracts are effective. Simple sedatives in small doses—phenobarbital or bromides—will at times relieve symptoms, and when headache and dizziness are relieved, the pressure may fall slightly. In crises of high pressure, nitrites or bleeding may afford temporary relief. Next in importance is the value of early recognition of the tendency of the individual patient toward the common (cardiac) problem or the less common renal termination. In the latter instance, the measures to be noted under nephritis may be applicable; in the ordinary patient they are not indicated. When frank cardiac disease develops, the mode of treatment is usually clear.

The therapy in the average case of hypertension without complications is one of the most common problems in medicine and it seems worth while to touch on it in some detail. Diet should be designed to cause loss of weight if the patient is obese (directed against potential cardiac complications), and possibly be made low in total salt content (restriction of free salt at the table). There is no proof that restriction of total protein intake is of any value, and the prohibition of so-called red meats in favor of lamb, fish and chicken is childish; the body is color-blind. There is no proof that condiments are in any way prejudicial to high blood pressure. Tobacco in moderate quantities will contribute to the patient's happiness, and so may alcohol in small amounts; neither will raise blood pressure. Simple laxatives, if needed, are preferable to strong purges. As in any other person with potential cardiac disease, violent exercise, such as tennis, squash, swimming, sculling and climbing, should be forbidden, but riding, walking and a moderate amount of golf are permitted. When fatigue is a prominent

symptom, a rest period of one and a half hours after the noon meal, and of one-half hour before and after the evening meal, may be helpful. A couch at the office may take the strain from the business man or executive. Vacations at more frequent intervals contribute much to the sense of well being, and may include sea trips, fishing, etc. Long automobile rides bring on the strain of driving in either front or back seat, and are best avoided. A sound night's sleep is essential, and the simple hypnotics—barbital, phenobarbital or chloral—may be employed. It is a paradox that many medical procedures, diagnostic or therapeutic, carry with them such ambivalent values; this is especially true of the estimation of blood pressure. The wise physician will avoid letting the patient with hypertension follow his pressure levels, and will perhaps avoid the determination entirely. Moreover, it is not necessary that the patient be under frequent observation; the monthly revisits to the doctor serve to maintain uncertainty and fear, both potent factors in elevating blood pressure. Some responsible person in the patient's family should be warned about symptoms or signs of importance, but it is well not to make watch-dogs of the family; their zeal will exceed their medical skill and usually their common sense. Save as indicated, drugs may be entirely omitted from the scheme of treatment.

NEPHRITIS

During the acute phase of hemorrhagic nephritis, one is commonly faced with the problem of edema, of nitrogen retention and of location and eradication, if possible, of the focus responsible. Edema is often, to some extent, a protective mechanism. In any case, diuresis by drugs is sharply contraindicated, since the basis for drug diuresis is renal irritation; neither custom nor the reports of the drug firm detail man should alter this course. Water should be allowed up to the limit of the capacity of the patient for excretion of urine, and usually can be forced. Starting at 1,500 cc. per day, it can be increased to 2,000, 2,500 or even 3,000 cc. if the volumes of urine maintain an approximate ratio of 1,000 cc. less than the intake. If there is nitrogen retention, it may be wise to set the dietary protein at a minimal level: 0.75 Gm. per kilogram of body weight, usually totaling from 40 to 60 Gm. a day. This quantity can be raised gradually if the nonprotein nitrogen does not increase. Liberal feeding of carbohydrate and fat is essential if protein is restricted. There is no merit to the diet of milk alone. Salt and all sodium compounds should be strictly excluded. As soon as the condition of the patient permits, infected teeth, sinuses or tonsils should be attacked surgically, with the usual reservation of several weeks between acute tonsillitis and surgical therapy. Until blood

has disappeared from the urine, as shown by ordinary tests at least, these patients should be kept in bed. Warm clothing and the utmost care of the skin are essential.

In the chronic stages, a good dietary is wise, with no special restriction of protein unless evidences of uremia or impending uremia are found. Inadequate protein feeding may help in depleting plasma proteins, resulting in an edematous phase. If edema or cardiac complications develop, salt and all sodium compounds should be withheld. In view of the tendency toward acidosis, the diet should contain liberal amounts of fruits and leafy vegetables, the alkaline ash of which will help to maintain body neutrality. Anemia may be treated with iron or by transfusion of blood. Since anemia usually means severe glomerular damage and faulty excretion of nitrogen, liver (the value of which in secondary anemias is less than that of iron) is barred. The frequent cardiac complications need much attention. In severe stages with threatened uremia, minimal protein feeding (0.66 Gm. per kilogram of body weight daily) and fluids forced in every way are indicated. Vigorous purgation may be necessary, and sedatives needed to control convulsive attacks.

In degenerative nephritis, a rigorous salt-free diet with ample protein (from 125 to 150 Gm.), to combat depleted plasma proteins, is the most effective measure against edema. Fluids may be restricted to 1,500 cc. daily. Small doses (from 0.065 to 0.130 Gm. daily) of thyroid extract may help in dispersing anasarca. Unless potassium salts are thought worth a trial, diuretics have no place in treatment. Foci in the nasal accessory sinuses especially should be eradicated, and transfusions of blood may be helpful. In view of the susceptibility of these patients to the pneumococcus, the utmost care in protecting them from colds and their sequelae is worth while.

In the arteriosclerotic varieties of nephritis, one must weigh the relative importance of cardiac and renal factors, and set the scheme of treatment accordingly. In general, cardiac problems will respond better than renal, and cardiac management will contribute more to the patient's comfort. In uremia, the principles outlined for hemorrhagic nephritis hold. Since the retinal changes depend on vascular degeneration and not on renal failure only, it is unlikely that vigorous purgation or similar measures will help rapidly failing vision.

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News and Notes

SOCIETY NEWS

International Association for Prevention of Blindness.—At 10 a. m., on April 17, 1933, at the Palace Hotel in Madrid, the International Association for the Prevention of Blindness will follow the program listed: 1. Address by the president, Professor de Lapersonne, on the activities of the association since its creation at The Hague in 1929. 2. Report by the vice president, Dr. Park Lewis, on behalf of the executive committee. 3. Election of members of the staff and the executive committee. 4. Reports of national committees. 5. Discussion of a scheme of international classification of causes of blindness. Members who wish to participate in this discussion are requested to send to the Secrétariat, 66, Boulevard St.-Michel, Paris, their names and a short (approximately twenty lines) summary of the proposed discussion. Discussions must not exceed ten minutes.

Acta ophthalmologica.—At the next Scandinavian Congress to be held in Stockholm during 1934, a new editorial staff will be elected for *Acta ophthalmologica,* which is now being conducted temporarily by Drs. Holm and Moller.

PERSONAL

To take the place of the late Professor Lundsgaard, Dr. H. Rönne has been appointed professor of ophthalmology at Copenhagen.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Conjunctiva

TUBERCULOSIS OF THE CONJUNCTIVA. A. V. DENTI, Ann. di ottal. e clin. ocul. **60**: 88 (Feb.) 1932.

Three cases are reported, and photographs and photomicrographs of material removed for biopsy are presented. All of the cases showed infiltration and papillary hypertrophy of the folds and bulbar conjunctiva. In the first two cases a diagnosis was easily made by positive skin tests (papillae arising on an area scarified and treated with tuberculin) and the finding of acid-fast bacilli in the biopsy material. In the third case, the skin test was negative, and bacilli were not found, but inoculation into guinea-pigs gave positive results as in the other two cases. In the first two cases the infection had apparently arisen from undoubted foci of pulmonary tuberculosis. In the third case no evidence of involvement elsewhere was present, and the infection was assumed to be primary in the conjunctiva. In all three cases roentgen treatment was successful in causing the lesions to heal, although one patient died soon afterward of tuberculous meningitis. (Bibliography.)

S. R. GIFFORD.

ANATOMIC PICTURE OF PIGMENTATION OF THE CONJUNCTIVA IN VITAMIN A DEFICIENCY. A. PILLAT, Arch. f. Ophth. **128**: 201 (April) 1932.

The clinical picture of pigmentation of the conjunctiva in vitamin A deficiency was described in a previous paper by the same author. Clinically the pigmentation is a valuable diagnostic sign of vitamin A deficiency in adult Chinese. Anatomically, however, it was found that the conjunctiva of every Chinese contains pigment. The difference between the normal and the pathologic pigmentation is not only quantitative but qualitative. In the conjunctiva of persons with vitamin A deficiency a new type of cells is found, namely, cells with a cubical or polygonal body, a long oval nucleus and dendritic processes. These cells become numerous and occur mainly in the deeper layers of the conjunctiva. They are real melanoblasts and take their origin from normal basal cells of the epithelial layer. The normal basal cell in the Chinese has a cap of pigment over the distal pole of the nucleus, at which point the pathologic formation of pigment starts. The cells of the other layers also form pigment. Extracellular pigment is never found. The formation of pigment seems to be a defense reaction of the living cell and not a function of the deteriorating cell. The corneal epithelium does not form pigment, which may be one of the reasons why the cornea so frequently undergoes severe changes (keratomalacia) in vitamin A defi-

ciency. Cells which normally contain keratohyaline particles like those of the conjunctiva bulbi form little pigment. The keratohyalin in itself seems to protect the cell sufficiently. The greatest degrees of pigmentation are found in the lower fornix and in the conjunctiva of the lower lid.

P. C. KRONFELD.

THE ETIOLOGY OF FOLLICULOSIS CONJUNCTIVAE. H. RIEGER, Arch. f. Ophth. **128:** 312 (April) 1932.

This paper contains further arguments and experimental data in favor of Lindner's assumption that Bacterium granulosis is the cause of folliculosis and not trachoma. Folliculosis is clinically easily differentiated from trachoma by the presence of transparent, very prominent follicles in an otherwise normal conjunctiva, which disappear spontaneously within from one to two years. The disease is infectious, is always bilateral and occurs mainly in younger people.

Four typical cases of this condition were used for bacteriologic studies. From each case bacteria could be grown. In the first case, further studies were impossible because the strain died very soon. In the second case, gram-negative bacilli were found, not at all similar to the bacterium of Noguchi. The bacteria grown from cases 3 and 4 closely resembled Noguchi's bacillus. When injected into the eyes of four Macacus rhesus monkeys, the cultures produced no definitely pathologic changes. When injected into one of Lindner's eyes, they produced a mild folliculosis which cleared up within one year. The author concludes that the bacillus derived from a patient with definite folliculosis behaves much like Noguchi's Bacterium granulosis and also produces folliculosis in man. He proposes the name Bacterium folliculosis for his strain or variety of Noguchi's bacterium.

P. C. KRONFELD.

Cornea and Sclera

EPISCLERITIS DUE TO ALLERGY. R. M. BALYEAT and H. J. RINKEL, J. A. M. A. **98:** 2054 (June 11) 1932.

A physician, 37 years of age, had for ten or twelve years attacks of episcleritis at intervals of five or six months. During the same period of time he had seasonal hay fever. He was given preseasonal pollen therapy of ragweed and pigweed. Foods to which he was specifically sensitive were removed from his diet, and he was advised concerning the possibility of feathers as a secondary factor in seasonal hay fever.

During the past year he has not suffered recurrent attacks of episcleritis except on one occasion, when he deliberately ate some of the foods to which he is specifically sensitive.

W. ZENTMAYER.

A FAMILY WITH GROENOUW'S DYSTROPHY. E. V. SRINIVASAN, Brit. J. Ophth. **16:** 296 (May) 1932.

The following scheme shows the members affected and the type of dystrophy. It is to be noted that the affected members were females, that in the three older ones the dystrophy was of the lattice type, in the

next two of the Groenouw type and in the two youngest of the superficial punctate type.

Name	R. (a widow)	N.		S.
Age	40	37		30
Variety	lattice-shaped	lattice-shaped		lattice-shaped
Vision	R.6/36 L.6/18	R.6/18 L.6/24		6/18 B.E.
Name (no issue)	C.	R.	N.	R.
Age	13	10	6	6
Variety	Groenouw's nodular	Groenouw's nodular	K.P. sup.	K.P. sup.
Vision	B.E. 6/6 pt.	B.E. 6/6 pt.	6/6 B.E.	6/6 B.E.

W. ZENTMAYER.

COLORED AREAS IN THE SCLEROTIC. C. H. USHER, Brit. J. Ophth. **16:** 671 (Nov.) 1932.

The author had the opportunity to reexamine six of nine patients with colored areas in the sclerotic after intervals ranging from six to twenty-one years. The color of the patches is described as purple, violet and lilac and slaty blue. The patches may be bilateral, but are usually monolateral. There may be one or more areas involving a larger part of the sclerotic, or only a small section. The patch may reach the corneal limbus anteriorly, or normal sclerotic may separate it from the limbus; the posterior margin is readily seen. Associated pigmentary conditions of the conjunctiva are not uncommon. In the author's series, four of the eyes showed low myopia; the remaining seven were hyperopic or emmetropic. In one instance a mother with melanosis of both eyes had a daughter with a dark patch in one eye.

Of the six cases that were reexamined after a period of years, the appearances had not changed in two cases, but definite changes had occurred in two other cases, in one as regards color and in the other as regards an increase in area. In the remaining two cases any change was in doubt. The results suggest that further observations are needed, and that measurements are necessary on first examinations in order to detect subsequent minor changes. Information is required regarding what occurs during the first two or three years of life.

W. ZENTMAYER.

THE EVOLUTION AND DIFFERENTIATION OF ECZEMATOUS AND TRACHOMATOUS PANNUS. I. GRIGORI and L. DIMITRU, Ann. di ottal. e clin. ocul. **60:** 81 (Feb.) 1932.

Based on a slit-lamp study of several hundred cases at the clinic in Jassy, Rumania, the authors describe a picture characteristic for each type of pannus. In eczematous pannus the vessels entering the cornea are derived chiefly from the scleral vessels and involve the deeper layers of the cornea. They advance toward the corneal center in small concentric circles, formed by an alternation of short corkscrew vessels with secondary plexuses that are derived from the spirochete-like vessels. These are extensions of ampulliform varices of the limbar plexus. During this process the cornea between the vessels remains clear. Infil-

trates develop, however, and the vessel arrangement is obliterated and replaced by one of an entirely different character, more superficial and at times difficult to distinguish from trachomatous pannus.

In trachoma the process is simpler, the vessels being derived only from the pericorneal plexus, which is supplied by episcleral vessels. Fine vessels from this pannus invade the superficial layers, anastomosing to form a series of loops which become smaller toward the corneal center. In the older cases one or more of these vessels enlarge, and their branches occupy most of the corneal surface to the exclusion of smaller vessels. Infiltration of the cornea is present from the beginning of the vascularization and proceeds with it. In later stages, a few vessels of deeper origin enter the cornea.

S. R. GIFFORD.

General Disease

NODULAR IRITIS IN DIABETES INSIPIDUS: REPORT OF A CASE. A. HOMMEL, Klin. Monatsbl. f. Augenh. **87:** 492 (Oct.) 1931.

Ocular complications have rarely been observed in diabetes insipidus. Cataracts, bitemporal or homonymous hemianopia, exophthalmos, ophthalmoplegia interna and paralyses of the ocular muscles have been reported in connection with diabetes insipidus. Hommel reports a case which presents some features similar to a case of Stoewer's. A man, aged 22, had suffered from frequent attacks of tonsillitis, some attacks of epilepsy, polyuria and polydipsia since childhood and fistulating glands of the neck. He complained of fatigue and fever when admitted to the hospital. The eyes, free from pathologic changes at that time, became inflamed soon afterward, finally requiring his transfer to the ophthalmic hospital. Several yellowish-gray nodules were found in the periphery of the iris of both eyes; also deposits on Descemet's membrane and floating opacities of the vitreous. Typical symptoms of iridocyclitis developed; the nodules increased to the size of lentils, but differed from tuberculous nodules. The Wassermann reaction was negative, tuberculosis could be ruled out and no etiologic infection could be detected. New nodules formed during the next month, but disappeared without leaving scars, while general recovery progressed. The disks remained slightly veiled for a brief period of time. No changes of the skull or sella turcica could be discovered by roentgenograms. No involvement of the hypophysis or endocrine dysfunction was detected.

K. L. STOLL.

Lens

RESULTS OBTAINED IN FORTY-FOUR CASES OF CATARACT IN WHICH OPERATION WAS PERFORMED BY THE METHOD OF JUNÈS. A. BUJADOUX, Ann. d'ocul. **169:** 545 (Aug.) 1932.

Statistics giving the results of cataract extraction are not published to any great extent in the medical literature. The method which Junès proposed in 1930 cannot help but interest practitioners. At the French Ophthalmological Society, May 13, 1930, Junès presented a paper, "The Best Corneal Incision in Operation for Senile Cataract: Vertical Keratotomy with Scissors."

The procedure is as follows: The incision is made at 2 o'clock for the left eye and at 7 and 8 for the right. It is placed exactly at the level of the limbus and the lance extends into the anterior chamber until the point reaches the pupillary border of the opposite side. It is then quietly withdrawn upward in the left eye and downward in the right. A lance with a base of 11 mm. is employed, and with it an incision occupying a little more than a third of the corneal circumference is obtained. The scissors are then introduced, and it is endeavored to push them just to the edge of the inferior extremity of the vertical meridian. The section is made with a single incision.

The details of the results obtained in forty-four cases are given and also the advantages claimed for this form of operation.

S. H. McKee.

THE PHYSICOCHEMICAL MECHANISM OF THE GENESIS OF THE CATARACT. P. WEINSTEIN, Klin. Monatsbl. f. Augenh. **87**: 393 (Sept.) 1931.

In an attempt to study the origin of cataract, physicochemical processes in the lens, in its capsule and also in the aqueous humor must be considered. In aging persons a diminution of cysteine, a derivative of glutathione, was found, in consequence of which the oxidative activity of the lens diminished. Hysteresis, i. e., dehydration of the colloids, involving the capsule of the lens, exposes the lens to toxic influences. The aqueous humor is of importance for the accumulation of substances that are detrimental to the lens, or for the promotion of a change of concentration of the hydrogen ions. The changes in the lens can be demonstrated by the nitroprussic reaction (NPR), devised by Reis and Arnold. It produces a purple discoloration of the lens, if combined with one drop of ammonia. Several authors found the result negative in hypermature cataracts, and partly positive in the clear portions of advancing cataracts.

Weinstein tested thirty-five extracted lenses with this method. The result was positive in all the capsules and in the cortex of ten immature cataracts, but negative in their nucleus; it was positive in the capsules and in several nuclei of phakosclerosis, positive also in two posterior cortical, one tetanic and one zonular cataract and in the capsule of a complicated cataract, which reacted in a negative manner. The result was negative in four morgagnian cataracts and in the lenses of ten hypermature cataracts, eight capsules of which gave a positive reaction. Twenty-seven of these cataracts were extracted in the capsule; they yielded positive reactions with the exception of two hypermature cataracts. Traumatic and toxic cataracts, as well as naphthalinic cataract, according to Jess, showed positive reactions in their clear and opaque portions. Weinstein obtained a similar result in one zonular and two posterior cortical cataracts; he stresses the fact that the reaction in senile cataracts, excepting in their capsule, was negative as a result of a biologic change in the lens, but positive in traumatic and toxic cataract, because the permeability of their capsule was damaged, so that opacification could take place by colloidochemical alterations.

K. L. Stoll.

ACUTE CATARACT WITH COMPLICATIONS: REPORT OF A CASE. H. ERGGELET, Klin. Monatsbl. f. Augenh. **87**: 825 (Dec.) 1931.

A slender, healthy looking woman, aged 29, showed thyreotoxicosis and a 30 per cent increase in metabolism; exophthalmic goiter of two years' duration was present. Vision in both eyes had decreased for about nine weeks without any known cause. The eyes were free from congestion, the palpebral fissures were wide and exophthalmos amounted to 12 mm. in the right eye and 13 mm. in the left. The upper lids stuck on looking downward; bilateral homonymous superficial opacities and reduced sensitivity of the corneas, atrophy and coarse vascularization of the iris were present. The pupils were wide, irregular and rigid. The anterior chambers were narrowed by strongly swollen cataracts. Intra-ocular tension was 18 mm. in the right and 20 mm. in the left eye. Perception of light, but faulty projection, was present in the right eye; motion of the hand was recognized and projection was uncertain in the left eye. Diabetes was absent; the blood sugar content was normal. The fundus was seen a few weeks prior to Erggelet's examination by another oculist, who found yellow discoloration of the disks, a venous pulse on slight digital pressure and contracted fields. A large raised area was observed near the disk and fresh linear hemorrhages below it. In addition to the general symptoms mentioned, there were slight excitement, weakened pulse of the upper extremities and increased blood pressure of the lower ones. The Wassermann test was negative. No explanation of this peculiar case was given.

K. L. STOLL.

UREA CONTENT IN THE NORMAL AND PATHOLOGIC CHANGES OF ANIMAL LENSES. SCHMERL, Klin. Wchnschr. **11**: 953 (May 28) 1932.

Because of the fact that the lens is rich in albuminoids, especially arginine (amino-guanidine-valerianic acid), the author was interested to know if urea was found in the normal lens and if there was any particular difference in amount between normal and cataractous lenses. Two rabbits killed after three days had had massage cataracts produced in one eye. Both normal and cataractous lenses were placed in 6 per cent xanthydrol solution and examined microscopically. In the lens capsules of both normal eyes a rich deposit of urea-xanthydrol crystals was found, while the lens nuclei also had the typical crystals but not in such abundance. In the cataractous lenses there were strikingly fewer crystals in both the capsules and the nuclei, and in the latter there were microscopic fields with no crystals. Thus the urea content of normal lens (also found in pigs' eyes) is proved, and the removal of urea in cataractous lenses is noted. An explanation of this process may be found in previous work by Schmerl. In beginning cataracts the acid content of the lens increases, while the sulphydrate body of the lens goes over to the aqueous. A similar observation has been made by Waldschmidt and Leitz in other albumin-splitting processes, and lately by Mothes in a study of the onion. Urea in the normal lens is evidence of albumin splitting under the activity of an arginase, and in the loss of urea in the cataractous lens there is a checking of this process in the lens by the loss of the SH radicle through an oxidation reduction reaction. Similar experiments are under way on human lenses. A bibliography is appended.

L. L. MAYER.

Methods of Examination

USE OF THE DIPLOSCOPE FOR THE MEASUREMENT OF HETEROPHORIA. BEYNE, Arch. d'opht. **49:** 379 (June) 1932.

The fact that artificial heterophoria seems to have a fixed value for a given optical instrument in each case has aroused some doubt in the author's mind in regard to the measurement of low degrees of heterophoria by means of prismatic apparatus. In this paper he describes a simple apparatus by means of which it is possible to measure the deviations in minutes.

S. B. MARLOW.

A NEW HEMIKINESIMETER. G. BRAUN, Klin. Monatsbl. f. Augenh. **87:** 441 (Oct.) 1931.

In the first chapter the author discusses the merits of hemikinesia, or hemianopic rigidity of the pupils, in localizing intracranial pathologic foci. While a number of leading physicians doubt the existence of this symptom for theoretical reasons, others consider it a decidedly important factor in differential diagnosis. Braun considers that not only [illegible] narrow pupils and reflex rigidity but also the lack of an accurately working apparatus for the examination are severe obstacles in the correct evaluation of hemikinesia.

The chief requirements for an exact examination are uniform intensity, distance and angle of incidence of the light used for stimulation, and a uniform term for the state of adaptation of the eye. Furthermore, isolated illumination of a limited, peripheral area of the retina, prevention of diffusion of the light and its reflection in the fundus and avoidance of simultaneous stimulation of the fovea are necessary. An instrument fulfilling all these requirements is the new hemikinesimeter, constructed after Elschnig's device in a manner which permits one to obtain a pupillomotor effect from any isolated spot in the retina. A detailed description of this instrument, built by Dr. Hartinger of the Zeiss Company, Jena, is given by Braun. Here the shortest possible interval of illumination can be obtained, diascleral light is excluded, and diffusion and reflection of the light, used for examination, are reduced to a minimum. With this instrument due consideration can be given to Hess' postulate and to divers values obtained in stimulating nasal or temporal retinal areas, and the graduated and exact focusing of eccentric portions of the retina in the horizontal and oblique meridians can be read easily. The results of examinations will be published later.

K. L. STOLL.

A NEW METHOD FOR MEASURING THE REPRESSIBILITY OF THE GLOBE IN THE ORBIT (PIESIMETRY). H. PLEGGE, Klin. Monatsbl. f. Augenh. **87:** 584 (Nov.) 1931.

Normal changes in the position of the globe in the orbit are caused by opening of the lids, stimulation of the sympathetic nerve, bending of the head and forced expiration, which produce proptosis. Retraction of the globe, in the shape of enophthalmos, follows closing of the lids, paralysis of the sympathetic nerve, retroflexion of the head and forced inspiration. Therefore, it is necessary, for obtaining correct measure-

ments of the repressibility of the globe, that the patient be placed so that the globe assumes "a position of rest." Maximal repressibility cannot be measured without endangering the safety of the eye. A weight of 50 Gm., amounting to a pressure of 100 Gm. on the globe, has been adopted as most suitable for taking measurements with the piesimeter. This instrument is constructed on the principles of that of Langenhan's, and in adherence to the observations made with Mangold's tonometer. The new apparatus is described in detail, and thirty-four measurements taken on twenty patients are given in tables. The average repressibility was 4.41 mm., as compared with 4.7 mm., the result obtained by Langenhan. A difference of from 0.1 to 0.3 mm., or 0.4 mm., in the eyes of the same person constituted the unavoidable latitude of error, which is of as little consequence as far as the result is concerned as are the age and the intra-ocular tension of the patient. Only very low tension may have some bearing on the results of the measurements. The piesimeter is valuable in differentiating the diagnosis between intra-orbital tumor and inflammation in the orbit, or between solid and vascular tumors; also in studying the progress of intra-orbital processes, exophthalmic goiter and disturbances of the ocular muscles.

K. L. STOLL.

TOPOGRAPHIC PHOTOGRAPHS OF THE RETINA FOR LOCALIZATION OF RUPTURES BEHIND THE EQUATOR OF THE EYEBALL. J. L. PAVIA, Klin. Monatsbl. f. Augenh. **87**: 657 (Nov.) 1931.

The picture obtained in the ophthalmoscope covers a radius of 5 mm. if the diameter of the nerve head measures 1.5 mm. Pictures taken on films of 4.5 by 6 cm. with the Nordenson-Zeiss apparatus show the vertical diameter of the optic disk to be from 5 to 7 mm. Therefore, the distance of ophthalmoscopic detail can be figured out in disk diameters, while the proportions of all photographic sectors remain constant. The author took panoramic pictures of detachments of the retina by piecing together pictures of adjoining sectors. Illustrations furnish stereoscopic views of the course of the same vessels before and after ignipuncture of a detachment of the retina.

K. L. STOLL.

Neurology

OBSERVATIONS ON THE ETIOLOGY AND SYMPTOMATOLOGY OF DISSEMINATED SCLEROSIS. W. J. ADIE, Brit. M. J. **2**: 997 (Dec. 3) 1932.

This is an important article on disseminated sclerosis, and there are many points which are of interest to ophthalmologists. The report is based on a study of two groups of cases: 70 cases of acute retrobulbar neuritis, seen at the Royal London Ophthalmic Hospital, and 118 cases of established disseminated sclerosis, seen elsewhere. Disseminated sclerosis is next to syphilis the most frequent disease of the nervous system. The attempts to isolate a specific organism or to transmit the disease have not succeeded. Knowledge of the clinical manifestations, however, is increasing rapidly. While thirty years ago the disease was characterized by nystagmus, intention-tremor and scanning speech, these signs are now commonly absent. The modern tendency is to emphasize an insidious onset, though acute retrobulbar neuritis is frequently stated

as the first manifestation. In discussing monosymptomatic onset, the first symptom was transitory ocular troubles (amblyopia, diplopia, etc.) in 40 per cent of the cases and transitory or variable motor or sensory disturbances in one or both lower limbs in 30 per cent. The age of onset is the third or fourth decade in 74 per cent of the cases. The value of nystagmus as an ocular symptom in differential diagnosis is limited, and it is frequently absent. Paresis of an ocular muscle sufficient to cause diplopia is very common, while squint, or a defect so gross as to be detectable by rough tests, is very rare. A persistent gross ocular palsy in a patient free from other symptoms is not suggestive of disseminated sclerosis. Transient diplopia is a common initial symptom, and may precede all other signs or symptoms by many years. Alterations in the pupils are rare, and the presence of defective pupillary reactions should cause doubt about the correctness of the diagnosis. Lesions in the optic nerve, chiasma and tracts are common, but permanent blindness or permanent severe loss of vision is rare. Mild transitory attacks of amblyopia are common.

Acute Retrobulbar Neuritis.—Of 118 cases, a clear history of a severe attack of retrobulbar neuritis was obtained in 41. In every instance it was an early symptom; it always appeared during the stage of the disease characterized by premonitory symptoms and remissions. Acute retrobulbar neuritis seemingly rarely occurs after the disease is firmly established. It is well known that retrobulbar neuritis may precede other symptoms of disseminated sclerosis by many years. In the author's cases the intervals varied from a few weeks or months up to ten, fourteen, nineteen, twenty and twenty-four years. The average was 6.8 years. The simultaneous disease of both eyes was not recorded in a single instance, and distinct acute attacks in sequence within a month are mentioned only once. Therefore, simultaneous or early consecutive attacks are very rare, and their occurrence should suggest some other form of inflammation of the optic nerves. The relation of acute retrobulbar neuritis to disseminated sclerosis is not sufficiently recognized in textbooks on neurology.

The author defines retrobulbar neuritis as being characterized by: (1) acute onset, (2) unilateral incidence, (3) central scotoma and (4) tendency to recovery. Some pain is almost always present at the onset. Visual failure is rapid rather than sudden, reaching its maximum in a few hours, or at most in a day or two; it is always more intense in the central areas of the field. Recovery begins within a week or two, and by the end of six weeks or two months, the recovery is often regarded by the patient as complete, though examination with a small colored object may reveal a residual defect. The ophthalmoscopic changes in acute stages are not marked and are rare. Intense swelling, the typical choked disk, if it occurs at all, is very rare in the unilateral type. It is a common feature in the bilateral cases. The author suggests that intense swelling in one or both eyes, or bilateral incidence of even mild visible changes with severe visual failure, indicates a different etiology.

As to the frequency of retrobulbar neuritis in disseminated sclerosis, the author makes the following statement: 1. Disseminated sclerosis is the only known common cause of acute unilateral retrobulbar neuritis as defined in this article. 2. Apart from certain rare allied

acute inflammatory diseases of the nervous system, it is the only cause. If these statements are accepted as approximating the truth, there will be at least less heard of hypothetic sinus disease and dental or tonsillar sepsis as possible causes, and the incidence of disseminated sclerosis as the cause will no longer be so variously estimated as from 20 to 80 per cent (Traquair: *Tr. Ophth. Soc. U. Kingdom* **1:** 351, 1930). The author believes that evidence of the presence of disseminated sclerosis can be obtained in over 30 per cent of the cases of unilateral retrobulbar neuritis in the acute stage, and that diagnosis can be made with a close approach to certainty in about 50 per cent. The fate of the cases in which the cause is uncertain can be given only if the patients can be kept in touch with sufficiently long. Fleischer followed up some of his patients and found that in 66 per cent disseminated sclerosis developed. In acute uncomplicated cases and in patients with established disseminated sclerosis giving a history of an acute attack of visual failure in one eye as their first symptom, the acute attack occurred generally between the ages of 21 and 30, and with a preponderance (64 per cent) in females rather than males. This age factor and sex preponderance in females suggest a common cause. Furthermore, the conclusion seems justified that females are more susceptible than males, and that acute retrobulbar neuritis is a more frequent initial manifestation in females than in males.

A. KNAPP.

OPHTHALMIC MIGRAINE. SIDLER, Schweiz. med. Wchnschr. **62:** 529 (May 28) 1932.

A case of migraine with unusual findings is reported. Typical ophthalmic migraine is found in persons of an angioneurotic nature and is thought by Rodella to be due to a spasm of the internal carotid, or at least of the ophthalmic artery or its branches. The second phase is the headache due to paralysis of the vessel and return of vision. The author's patient had frequent attacks of endocarditis. Ten years before, he had an embolus of the central artery of the right eye, absolute central scotoma of 25 degrees and a pale nerve with thinned arteries. During the attack there was a rapid diminution of the vision of the left eye from 1.3 to 0, which lasted from ten minutes to three hours. The ophthalmoscopic picture of the left eye was normal. The interesting part is that an attack with diminution of vision and subsequent headache could be induced by light pressure on the left eyeball. When the fundus was viewed after this procedure the disk was snow-white, and the vessels, especially the arteries, were completely collapsed. With release the fundus returned to normal and the headache persisted. The author believes this to be a case in which the most peripheral vessels showed the changes usually attributed to central arteries.

L. L. MAYER.

THE MEANING OF HIPPUS IN THE DIAGNOSIS OF MULTIPLE SCLEROSIS. F. HERZOG, Med. Klin. **28:** 1705 (Dec. 2) 1932.

For the past twenty years the author has noted this symptom in multiple sclerosis and other diseases of the nervous system. He reports

three cases seen in the past two years in which hippus was a sign which made the diagnosis. Previous cases seen by the author coincide with the three cases outlined. The features which distinguishes this hippus from that found in some normal eyes are that the excursions are greater than 1 mm., with long pauses between reactions, and also it does not occur in both eyes at the same time or degree. The movement of the pupil did not occur in connection with sensory or psychic stimuli and were greatest when the patient gazed at a distance in a poorly lighted room. Hippus was also found in cases of progressive paralysis, tabes, meningitis, oculomotor paralysis, tumor of the brain, intracranial hemorrhage, dementia praecox, hemiplegia, encephalitis endemica, chorea, poisoning caused by barbital, epileptic and epileptiform seizures, in trauma, in neurasthenia, and at times in apparently normal patients. In disseminated syphilis of the nervous system, hippus was not demonstrable. In all the aforementioned conditions, hippus occurred more infrequently than in multiple sclerosis. Damsch also believes that the sign occurs much earlier in multiple sclerosis and may be the only cerebral symptom. The author explains the greater frequency of hippus in multiple sclerosis by the fact that not only the nuclei but also the nerve fibers of the sphincter and dilator are probably affected in this disease.

There is a bibliography. L. L. MAYER.

Orbit, Eyeball and Accessory Sinuses

A CASE OF MUCOCELE OF THE MAXILLARY SINUS AFFECTING THE OCULAR GLOBE. Y. SHOJI, Ann. d'ocul. **169**: 267 (April) 1932.

M. S., a man, aged 45, who acquired syphilis at 17 and, three years before the present observations, submitted to an operation for nasal empyema, noticed a certain amount of irritation about the left lower lid four months previous to examination. This lasted about three or four days, but since that time the region of the lower lid and cheek had progressively enlarged. He complained of binocular diplopia. Examination showed a swelling about the inferior part of the left lower lid. The tumor, of elastic consistence on the nasal side and of bony consistence on the temporal, occupied nearly the whole length of the lid and measured 18 mm. from above down. The left globe was displaced above and to the left, and the ocular movements downward were limited.

Under procaine hydrochloride anesthesia an incision was made over this mass, and a fibrous growth was found on the anterior surface of the maxillary sinus. On perforation of this area, a brown liquid escaped. After ablation of the tumor, one could see the whole cavity of the maxillary sinus. Convalescence followed in one week's time.

Cases of mucocele of the maxillary sinus have rarely been observed in ophthalmology. The mucocele enlarges the cavity and may cause inflammation of the retina or inflammation and atrophy of the optic nerve. The deviation of the ocular globe is the chief ocular symptom, which disappears on the opening and evacuation of the cavity.

S. H. MCKEE.

The Pupil

SIMULTANEOUS INTERNAL AND EXTERNAL STIMULATION OF THE IRIS BY ADRENIN. T. SCHLOSSBERG, Am. J. Physiol. **102:** 71, 1932.

Epinephrine instilled into the eyes of cats causes no dilatation of the pupil until the iris has been sensitized by removal of the superior cervical ganglion. Previous investigators have noted that when an animal whose superior cervical ganglion has been removed is excited the pupil on this side dilates more than the control. This has been attributed to the action of epinephrine circulating in the blood as a result of the excitement.

In the present experiments the superior cervical ganglions of cats were removed aseptically on both sides. At varying periods after the operations the animals were artificially angered (shown a dog). Under these circumstances there appeared a strong dilatation of the pupils which receded sharply a few seconds after the end of the stimulation, frequently leaving a more moderate widening which disappeared gradually after several minutes. The latter effect was more marked the stronger the emotional excitement.

Fifteen days after removing the cervical sympathetic ganglions an instillation of epinephrine produced a definite dilatation of the pupil. If epinephrine was instilled in one eye at this time and the cat was excited emotionally, the pupil on the side of the instillation showed a more intense and lasting dilatation.

It is known that when the superior cervical sympathetic is removed the iris is still innervated by the third nerve, and the instantaneous dilatation of the denervated pupil in emotional excitement is probably due to central inhibition of the tonically active constrictor impulses. The author believes that the smaller dilatation persisting after the emotional stimulation has ceased is due to epinephrine circulating in the blood.

F. H. ADLER.

Physiology

AN UNSUCCESSFUL ATTEMPT TO DEMONSTRATE HUMORAL ACTION OF "VAGUS SUBSTANCE" IN THE CIRCULATING BLOOD. N. E. FREEMAN, R. A. PHILLIPS and W. B. CANNON, Am. J. Physiol. **98:** 435, 1931.

The authors were unable to demonstrate the presence of "vagus substance" in the circulating blood of animals, although a variety of test organs was employed, some of which were sensitized by previous denervation, i. e., denervation of the iris by removal of the ciliary ganglion and the superior cervical ganglion. (This article is abstracted because of the numerous reports in recent German ophthalmologic literature of the presence of a "vagus substance" in the circulating blood, the eye being used as a test organ.)

F. H. ADLER.

Refraction and Accommodation

SCHOOL MYOPIA. A SORSBY, Brit. J. Ophth. **16:** 217 (April) 1932.

This is an excellent review of the modern conception of the myopic question. The author says that as much as recent work has clarified

the issue, it cannot be said that all established factors are covered by the more modern views. One bed-rock fact, which recent work seems to ignore in its stressing of the essential unity of all spherical errors, is the vastly different behavior of the hypermetropic and myopic eye during school life. It may well be that the progressive hypermetropic and the stationary myopic eye are but the beginning and the end-stages of that progressive process seen in myopia. It cannot be sufficiently emphasized that myopia develops during school life and particularly after the age when the growth of the eye is regarded as being complete —the age of 8 years.

Most school myopia becomes magnified at about the age of 12 years. If the growth of the eye has ceased by then, why should there be in a certain proportion of children a readjustment of axial length, or the position or strength of the refracting media? This can hardly be a physiologic process if it affects only a small percentage of children.

It is unlikely that we shall solve the problem of myopia without intensive study in its breeding ground—the school.

W. ZENTMAYER.

Retina and Optic Nerve

SPONTANEOUS CURE OF DETACHMENT OF THE RETINA. WHAT ARE THE CHANCES OF FUNCTIONAL RECOVERY OF A DETACHED RETINA? L. WEEKERS and R. HUBIN, Arch. d'opht. **49:** 65 (Feb.) 1932.

The authors describe two cases of detachment of the retina in which a perfect spontaneous cure resulted, from the point of view that no trace of the detachment could be found. In one case a considerable return of function took place, whereas in the second there was none. These observations lead the authors to point out the necessity of some means to determine the visual capacity of a detached retina, for it is useless to attempt an anatomic cure by operative procedures when return of function is impossible. While the age of the detachment is important, it is not the only consideration, for the rate of degeneration varies in different instances. Hence the necessity of the separate study of each case. The authors believe that operation should not be delayed too long if the detachment is localized, as such cases seem to offer the best prognosis, though not invariably so. It is their opinion, however, that intervention is not so urgent that an operation should be a precipitate one. Before undertaking it, one should make a careful search for some general cause and sometimes try appropriate general treatment. When the indications for operation are clear, it is preferable to intervene with the least possible delay.

S. B. MARLOW.

THREE CASES OF EMBOLISM OF THE CENTRAL RETINAL ARTERY: THE RÔLE OF SUPERADDED ARTERIAL SPASM. VILLARD, DEJEAN and TEMPLE, Arch. d'opht. **49:** 72 (Feb.) 1932.

The three cases described by the authors presented the classic picture of embolism of the central retinal artery. They believe that it would be more appropriate to use the term thrombus, as it is not a question of a cardiac embolus suddenly blocking a vessel but rather of an arteriosclerotic lesion with hypertension. Pathologic study has shown that the

central retinal artery is subject to atheromatous changes from which proliferative endarteritis may arise. The fact that in some cases partial return of function occurs is in favor of spasm of the vessel wall and against complete obstruction by an embolus. The authors refer to the recent reports by many different observers which have appeared in the literature, and especially mention the work which has been done by Coppez and Brenner and by Redslob to prove the existence of vasomotor nerve fibers in the retinal vessels. A comparison is made between this condition in the eye and Raynaud's disease. As a result of the study of their three cases the authors believe that it is possible to distinguish between those cases in which the arterial lesion predominates and those in which spasm is the important factor. No improvement is to be expected in the first group, whereas in the second, if the case is seen early enough, considerable recovery can be hoped for.

S. B. MARLOW.

THE MCCLURE-ALDRICH TEST AND THE FUNCTION OF THE CAPILLARIES IN IDIOPATHIC RETINAL DETACHMENT. A. FERRARI, Arch. di ottal. **39:** 279 (June) 1932.

The McClure-Aldrich test depends on the time required for the absorption of 0.2 cc. of physiologic solution of sodium chloride injected intradermally. This and wheal formation after a similar injection of histamine were noted in five cases of idiopathic detachment. The absorption of fluid was distinctly delayed in all cases, as was the wheal formation after histamine in four of the five cases. All showed increased venous pressure and abnormalities of renal function, as indicated by the concentration test. Such indications of abnormal fluid exchange in the tissue, the author believes, may be of significance in retinal detachment, although myopia is a predisposing cause in most cases.

S. GIFFORD.

TREATMENT OF RETINAL DETACHMENT. SOURDILLE, Arch. de oftal. hispano-am. **32:** 233 (May) 1932.

After reference to Gonin's theory, work and results, the author points out that his own method of treatment is based on a pathogenic conception which differs radically from that which constitutes the basis of Gonin's. He considers retinal detachment as primarily due to a defect in the union of the two leaves of the retina, and that the retinal rupture simply indicates a severe lesion at the site of its occurrence. The detachment produced is maintained and aggravated by a liquid exudate, mostly proceeding from the choroid where there is no rupture, and to a mixture of this with soft vitreous where this exists. The problems to solve, therefore, are: 1. The production of a readhesion of the retina to its pigment epithelium. 2. The induction, in the choroid and retina, of an adhesive inflammation sufficient to bring about an adhesion of these two membranes without, however, being destructive enough to produce anatomic lesions that may affect vision. The author obtains this by means of an evacuation of the subretinal fluid with multiple scleral punctures and by perforation of the retina.

The author describes his operative measures in detail. His method consists essentially in multiple scleral punctures at the site of the detachment with a Graefe knife, followed by subconjunctival injections of a 1:1,000 solution of mercuric cyanide.

In cases in which the detachment is limited and flat or in which the first procedure has failed, he substitutes the galvanocautery, which is here used as a means both of puncture and of producing a choroidal irritation.

After these procedures it is important to maintain immobility of the head for eight days in a position favorable for the retinal reattachment. Sourdille prescribes a postoperative fluid diet, bandages both eyes at first and keeps the patient in bed for from three to six weeks. His results in 169 cases are: 71 cures and 6 improvements. In 65 cases the knife was used, and in 24 the galvanocautery. In 43 cases only one operation was necessary; in 26, two; in 14, three or more. C. E. FINLAY.

VESSEL SHADOWS: A NEW SYMPTOM OF RETINAL DETACHMENT. O. AUST, Arch. f. Ophth. **129:** 17 (Sept.) 1932.

In three cases of retinal detachment and in one case of retinitis albescens, shadows of the vessels could be observed as dark lines, running parallel to the retinal vessels and changing their relative positions if the direction of the incident light rays was altered. The physical and anatomic conditions under which these lines become visible are discussed. They indicate an abnormally long distance between retinal vessels and pigment epithelium or, in other words, a shallow retinal detachment. They are visible only if the retina is highly transparent. The most favorable physical conditions for the detection of the shadows are: a very small ("point"-shaped) light source located within the anterior focal length of the eye and a large angle between the axis of the incident rays and the observer. The electric ophthalmoscope of May and the large Gullstrand ophthalmoscope fulfil those requirements.

In two cases, the shadows were noted after chemical cauterization with potassium hydroxide had been done. The shallow detachment was located posteriorly to the row of cauterized areas.

No tear could be found in the detached areas. The author believes that the shadows of the vessels in those cases indicate tangential traction on the retina exercised by the shrinking choroid. It seemed to be one of the main advantages of the chemical cauterization that new traction was rarely produced by the operative procedure or its sequellae. The cases with vessel shadows, however, suggest the presence of a shrinking process in the anterior choroid apparently produced by the after-effects of the extensive application of the caustic agent. P. C. KRONFELD.

ANGIOID STREAKS AND PSEUDOXANTHOMA ELASTICUM OF THE SKIN: REPORT OF A CASE. F. Poos, Klin. Monatsbl. f. Augenh. **87:** 734 (Dec.) 1931.

About sixty-five cases are known in the literature of the simultaneous occurrence of streaks in the retina, resembling blood vessels—so-called angioid streaks—and pseudoxanthoma of the skin. Both eyes are usually affected, showing more or less concentric curves, connected by anas-

tomoses, at a certain distance from the disks. Other streaks, occasionally running in a radial direction, stop at the equator of the globe, leaving the periphery of the fundus intact. No connections with the retinal vessels have been found. The streaks appear to be located between the retina and the choroid; their color varies from red to dark brown, or they resemble bright red vessels lined with bright seams of varying width, occasionally interrupted. Again the streaks may present the appearance of striated retinitis and extend into the extreme periphery.

Poos reports a case in an otherwise healthy man, aged 32, with an unimportant family history. Brownish-red concentric streaks could be seen surrounding both disks, some of them presenting typical spindle-shaped enlargements, and also anastomosing and radial streaks, which were accompanied by bright seams. All these streaks were crossed by the retinal vessels, and their substratum had a dark gray hue. The posterior region of the fundus appeared grayish red in comparison with the periphery. The centers of the retinas were intact, with the exception of a small white spot in the right eye. The vision was normal in both eyes. In the region of both sternocleidomastoid muscles dark discolorations were observed, with slightly raised, brownish-yellow areas. Histologic examination of an excised piece of the skin showed degeneration of the elastic tissue of the cutis, so-called elastoktasis or elastorrhexis, while the epidermis, the papillary body and the elastic tissue of the outer layers of the skin were normal. Angioid streaks and pseudoxanthoma elasticum may occur simultaneously in several members of a family, suggesting constitutional disease. The intra-ocular changes are brought on by brittleness of the elastic lamina. Decreasing elasticity of the glassy membrane coupled with the mechanical action of intraocular tension and that of the ocular muscles, may cause traumatic rents and dehiscences, comparable to the elastorrhexis of the skin over the joints. Hemorrhages, frequently observed in angioid streaks, may occur as a result of the close connection between the elastic lamina and the choriocapillary layer.

The genesis of the typical changes in the fundus cannot be explained, although minor contusions preceded their formation in two cases, described by Marchesani and Wirz. These authors consider the region between the retina and choroid a typical location for angioid streaks. The disease has a progressive and chronic character; it may lead to amaurosis which, in severe cases, can be prevented as rarely as the pseudoxanthoma can be cured.

K. L. Stoll.

The Reason for Failures in the Operative Procedures for Detached Retina. R. Bergmeister, Wien. klin. Wchnschr. **45:** 613 (May 13) 1932.

That the work of Gonin is an advance in the treatment of detachment of the retina there is no doubt; however, the author has tried to study some of the reasons for failure in such cases by investigating certain of these patients. The outstanding factor seems to be a hemorrhage into the globe. This appears to be due to injury to a vorticose vein during the operation. Patients with a high blood pressure, even in youth, are prone to hemorrhage and thrombosis. Cases are cited in which a tuberculous infection was in all probability the etiologic factor

in causing the separation of choroid and retina. Bergmeister demonstrates the importance of determining such a predisposing disease. Essential hypertension is also a contraindication to the operative treatment of detachment of the retina. The author repeats the axioms of Axenfeld, who made every effort to find other ocular changes besides the detachment, and stresses the fact that Elschnig prefers to wait at least six months before undertaking an operation. Cases are outlined to show the value of palliative measures, including the subconjunctival injection of saline solution. Four eyes treated with the Guist potassium hydroxide stick are recorded, in which the inability of the operator to control the alkali had harmful effects. The author disagrees with Lindner that the Guist method is an advance over that of Gonin because of the fact that the uncontrolled chemical may cause more damage than has already occurred. A bibliography is included. L. L. MAYER.

Trachoma

RESEARCH ON THE BACTERIUM GRANULOSIS OF NOGUCHI. C. TRAPESONTZEWA, Rev. internat. du trachome **9:** 173 (Oct.) 1932.

Not only the bacterium of Noguchi but that of Stépanowa, which has quite different cultural characteristics, has given positive results on inoculation in the rhesus monkey. Mikaélian found that his sample of B. granulosis could be transplanted on ordinary mediums, but the author's investigation demonstrated that he was not dealing with a pure culture. B. granulosis reacts variously with the Gram stain: Generally, the long forms are gram-positive and the short forms gram-negative. B. granulosis in symbiosis with a diplococcus of the author's (bearing no etiologic relation to trachoma) gives a much better growth on coagulated serum, and then the Gram stain is regularly negative. Agglutination tests with B. granulosis, tested with serum from trachomatous or nontrachomatous patients, are invariably negative. The etiologic rôle of B. granulosis must be still considered dubious; more experimental work, especially on man, is needed. J. E. LEBENSOHN.

CONSIDERATIONS CONCERNING THE PATHOGENICITY OF TRACHOMA. DELANOË, Rev. internat. du trachome **9:** 179 (Oct.) 1932.

Delanoë finds a close relationship between trachoma and tuberculosis in the type of lesions present, their course and reactions.

J. E. LEBENSOHN.

Tumors

PRIMARY NONPIGMENTED SARCOMA OF THE LID. C. GABRIELIDES, Ann. d'ocul. **169:** 593 (Aug.) 1932.

Primary sarcoma of the lid is a tumor rarely seen. Often it appears in the orbit itself or in the neighboring skin of the lids with secondary development in the tissue of the latter.

Reports of primary sarcoma of the lids are rare in the literature. Gabrielides reports a case of involvement of the left lower lid in a man

of 50 years who had neither hereditary nor acquired disease. The growth appeared spontaneously without any predisposing cause, and developed rapidly. An excellent therapeutic result was obtained by means of x-ray therapy, but three months and a half later the patient died from metastasis to the liver.

Details of the case and therapy are given, and also a historical account of former reports beginning with Desmarres, in 1851, Mackenzie, in 1856, and Deval, in 1862. There are two illustrations.

S. H. McKee.

Epibulbar Epithelioma. F. Mura, Ann. di ottal. e clin. ocul. **60:** 260 (April-May) 1932.

Three cases are reported in which recurrences of an epibulbar tumor required enucleation with evisceration of the orbit in two cases. In all cases, the cornea was infiltrated with epitheliomatous tissue, but the inner membranes were not involved.

S. R. Gifford.

Visual Tracts and Fields

Correlative Cortical Changes in the Brain of the Monocular Blind. L. Horn and M. Helfand, Wien. klin. Wchnschr. **45:** 1309 (Oct. 21) 1932.

In the examination of the brain of a deaf and dumb person changes were found by one of the authors under Professor Economo's direction. It was found that the most violent changes instead of being in the Heschl region (angular gyri), the auditory cortex in a narrow sense, where one might expect such changes, were in the so-called planum temporale, the cortical field behind the Heschl area. The planum temporale, however, is not the field of projection of a sensory region, but a field with indirect connections with the auditory region; so the severe changes in this province are known as correlative changes. With these ideas in mind, the authors were eager to investigate the cytologic structure of the brain in persons with monocular blindness. The article is the report of such a condition. The brain was obtained from a 52 year old patient who died in 1931, having for ten years had blindness of the right eye due to an ulcer serpens of the cornea. The findings are given in detail, but it is sufficient to say that in the projection fields of both sides of the optic pathways there were no changes; however, in the left hemisphere (contralateral side), of the angular gyri, the number and size of the cells were greatly decreased. Thus the authors conclude that these two conditions are similar, and such an addition is of interest in the study of cellular changes in the brain.

L. L. Mayer.

Therapeutics

Tuberculin in Ophthalmology. F. A. Williamson-Noble, Brit. J. Ophth. **16:** 285 (May) 1932.

When in obstinate chronic inflammatory conditions of the eye exhaustive search for the etiologic factor has failed, it has been the custom of the author to "try tuberculin." The object of the paper is to

try and give some experimental explanation for the success of this reagent in cases that are obviously not tuberculous and to give some clinical experience. In his efforts to attain the final object, he cites Wright's experiments which indicate that there is a certain dose of antigen that produces the optimum immunity response, and from further experiments he recommends that when the nature of the infecting agent cannot be determined one should not allow convictions that immunity is specific to hold one back from inoculating minimal doses of tuberculin B E, or in cases associated with rheumatoid changes, a vaccine of intestinal streptococci.

The author states his theoretical rationale for preferring the basilary emulsion.

He believes that some of the chronic eye diseases are instances of allergy, and as the secret of desensitization is to give small doses, his practice is to start with 1/500,000 mg. of B E and work the doses up slowly.

He cites an instance of disciform keratitis, with keratotic precipitates, vitreous opacities and a healed focus of retinochoroiditis, without clinical evidence of tuberculosis, in which the active lesions disappeared and vision was restored to 6/6.

A case of scleritis, one of recurrent iritis, one of chronic iritis and one of epithelial ulcer, developing later a disciform type of deep keratitis, responded favorably to tuberculin.

It seems to the author that there are two questions to be decided:

1. Are a certain number of cases of tuberculosis being missed because the lesion in the eye is falsely attributed to some other cause, and the true diagnosis not found until tuberculin is administered?

2. Has tuberculin a definitely nonspecific immunizing effect against organisms other than the tubercle bacillus and thus brings about a cure?

W. ZENTMAYER.

Society Transactions

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ALGERNON B. REESE, M.D., *Secretary*

Nov. 21, 1932

MARK J. SCHOENBERG, M.D., *Chairman*

A CASE OF LAGOPHTHALMOS RELIEVED BY A NEW METHOD. DR. ISADORE GOLDSTEIN.

This paper will be published in full in a later issue of the ARCHIVES.

A CASE OF NODULAR CORNEAL OPACITY (GROENOUW). DR. WENDELL L. HUGHES.

In 1890 Groenouw described the first recorded case of nodular keratitis, similar to the one presented here. In 1917 he collected the records of one hundred cases reported since the first case. In the majority, similar lesions were found in immediate relatives. In one case the genealogy was traced for seven generations, sixteen cases being found. The youngest patient in the cases recorded was 14 years of age. The onset of the condition is after puberty, and although it may be frequently stationary for long periods, it is usually slowly progressive. In the late stages, the cornea becomes opaque, with periods of inflammatory reaction. There have been several cases reported in which microscopic sections were described. The white nodular spots are composed of a hyalin-like substance. In the early stages they are entirely confined to the stroma in its superficial half, but in the later stages there is a degeneration of Bowman's membrane, causing nodular elevation, with thinning and breaking down of the epithelium. Vascularization of the cornea is a late manifestation.

In the case reported there is no disturbance of the blood vessels at the limbus, and no vessels are visible in the cornea. Vision has been poor for from ten to fifteen years, and at present is: right eye, 20/50; left eye, 20/50, unimproved by lenses. Ocular examination gave entirely negative results except for the corneas, which showed numerous thin white opacities. A general physical examination, including a chemical analysis of the blood and a Wassermann test, yielded negative results. The patient's father is dead, but there is a history of his having had trouble with his eyes for many years. The patient has two brothers and two sisters. Only one brother came for an examination, and his eyes appeared normal on slit-lamp inspection. The patient's daughter, who is 8 years of age, shows no signs of ocular disturbance.

THE TECHNIC OF FITTING CONTACT GLASSES. DR. LEGRAND H. HARDY.

The difficulties which arise in an endeavor to fit contact lenses properly may be referred to three general sources: the patient, the physician and the appliances.

Squeezing of the eyelids, usually the result of nervousness, takes place not only during the insertion of the glass but afterward. The condition may be controlled by reassurance and by a mild sedative such as sodium amytal combined with local anesthesia produced by 0.5 per cent *p*-butylaminobenzoyl *di*-methyl-aminoethanol hydrochloride. Small apertures occasionally present difficulties, particularly to the insertion of the larger Mueller glasses. On the other hand, paresis of the orbicularis muscle occasionally makes it difficult for the patient to retain a glass in the proper position. The pressure of trephine or iridectomy scars at the limbus may require marked alteration in the size and shape, or even the type, of lens used. Before contact lenses are to be used one should make every effort to clear up marginal blepharitis or catarrhal conjunctivitis.

Sympathy, patience, kindliness and imagination are essential in the physician. He may best acquire the proper attitude from inserting a glass into his own eye and noting the difficulties involved. The smoothness and facility with which the contact lenses are applied contribute greatly to the success of the trial. Manual dexterity is an important factor in success.

Difficulties caused by appliances include that referable to the glass itself and that caused by the applicators used to insert it. The former may be visual, due to an ametropia that is created, or insufficiently corrected, by the glass, or physical, due to the discomfort and pain resulting from an improperly fitted glass. The visual difficulty is concerned mainly with the corneal segment of the glass; the physical, with the proper scleral fit. It is of the greatest importance that the scleral portions of the lens be properly fitted if comfort is to result, and this is largely a method of trial and error. Close observation with the loupe and oblique illumination in conjunction with the use of fluorescein yield valuable information. The applicators used depend on the personal preferences of the physician, who may select from seven useful types. At the first trial, an estimation of the optically correct lens is made on the basis of the refraction or the ophthalmometer reading in the case of simple ametropia, or from direct inspection of the eye in the case of keratoconus. The lens is inserted, and the patient's manifest refraction with the lens in situ is measured. From the resulting data the lens that is most likely to give the best optical correction may quickly be determined either from tables or from graphs prepared for this purpose. Such tables or graphs have been prepared by Hartinger of the Zeiss works and by Siegrist, Heine and Feinbloom. All of them require certain assumptions as a basis for calculations, and hence are only approximations.

DISCUSSION

DR. CLYDE E. MCDANNALD: I want to speak of one interesting thing in connection with a case of keratoconus. The patient stated that while attending a motion picture two days previously, she suddenly experi-

enced blurring of vision. Examination with the slit-lamp showed that she had a conical cornea with an acute rupture of Descemet's membrane in the left eye. The entire cornea was edematous and milky in appearance. Vision was reduced to perception of hand movements. I performed a paracentesis and used 2 per cent pilocarpine with a pressure bandage for about five weeks. The left cornea cleared, and healed with very little scarring, but no attempt has been made to apply a contact glass.

The right cornea was conical with 18/200 vision, improving to 20/30, minus, with a contact glass.

DR. W. S. KNIGHTON: I presented two patients with keratoconus who have been wearing contact lenses for twelve and seventeen hours a day, respectively, with good results. At the New York Eye and Ear Infirmary, where contact lenses are tried on all sorts of clinical patients, my poorest results have been in patients with a high degree of myopia.

DR. OLGA SITCHEVSKA: I have had good results with the Mueller glasses in several patients. They learned to insert and remove the glasses at the first visit to my office. I should like to ask Dr. Hardy whether he has used the sclerokeratometer, an instrument invented last year in Germany, for measuring the corneal and scleral curvatures.

DR. L. G. H. HARDY: I have met the difficulty that Dr. Knighton describes in fitting contact lenses to highly myopic patients. I am afraid that in these cases the lens at times touches the cornea, and this the Zeiss glass must not be permitted to do. The indicated remedy is to utilize a lens with sufficient curvature and depth to clear the cornea. One is then limited, in optical correction, by the ability of the optician to make compensatory adjustments of the anterior surface refraction—in some cases this may be as high as minus 15 diopters.

I have never seen or used the instrument that Dr. Sitchevska mentioned. A keratometer and sclerometer have been used experimentally to make physical measurements of the curvatures of these surfaces. Similar in action to the well known Geneva lens measure, fine rods passing through a plate impinge on the corneal and scleral surfaces, and the relative positions of these rods are calibrated. I know of no optical means of performing this service, and as far as I know, the mechanically measuring instruments have never been produced commercially.

ASSOCIATION OF SCLEROSIS OF THE CEREBRAL BASAL VESSELS WITH OPTIC ATROPHY AND CUPPING. DR. ARNOLD KNAPP.

This paper appeared in full in the ARCHIVES in November, 1932 (**8**: 637).

DISCUSSION

DR. SOLOMON FINEMAN: In attempting to demonstrate calcification of the basal vessels, it is extremely important to obtain the finest detail that is technically possible in a roentgenogram. One is dealing with very fine calcifications within the cavity of the skull, and this fine calcification must be shown on "flat" films through two thick layers of bone, namely, the plates of the skull. For this reason it is always best to make stereoscopic films of the skull, so as to enable one to locate with greater certainty the position of these finely calcified bodies somewhere near the center of the skull. If the roentgenograms are too thin, they

do not show the granular fine structure of the skull bones, and the calcifications are lost. Also if the penetrability of the roentgen rays is too great, these fine calcifications are lost because of what is commonly known as the "burning out of the tissues." In this type of work if the technic tried is found to be wrong, the procedure must be repeated until the required fine detail is obtained. That is the first important point.

Second, on the single flat film, one may see various shadows superimposed over the sella or in close proximity to the sella; these do not in reality represent calcification of the basal vessels. When viewed stereoscopically, the shadows are recognizable as calcifications along the lateral skull walls, or unusually prominent ridging of these walls.

Third, one must differentiate calcification of the basal vessels from calcification of the suprasellar membrane stretched between the clinoid processes; also from calcifications which occasionally occur within the pituitary gland, which are usually rather irregular in contour. One should see quite clearly in the roentgenograms either a single curved shadow running across the base of the sella, representing presumably a heavily calcified carotid, or a double linear shadow representing perhaps a lesser degree of calcification within the walls of the internal carotid, observed tangentially.

The fourth point of importance is not to rely too much on a single lateral view, even if stereoscopic, because here again one may readily fall into error. These calcified vessels should be demonstrated, if possible, in several different views, namely, laterally, postero-anteriorly and cephalocaudally. On the postero-anterior views they appear in the mesial margins of the supra-orbital fissures. A heavily calcified carotid may appear in the form of a signet ring superimposed over the ethmoid regions. In the cephalocaudal view, designed for exposure of the sphenoids, one attempts to visualize these calcifications along the sides of the sphenoid sinuses. In this way, by means of stereoscopic postero-anterior and cephalocaudal views, one attempts to trace the same shadow and to determine whether the shadow corresponds in size, position and contour to a calcified basal vessel.

Finally, one must differentiate calcifications of the basal vessels from aneurysms of the vessels of the circle of Willis. These aneurysmal sacs may show considerable dilatation, and the calcification of their walls may be even less pronounced than in the walls of the basal vessels of normal caliber. The calcific deposits in aneurysms of the basal vessels appear as faint semilunar striations of flakelike deposits usually located rather high above the sella.

As to the age period in which calcifications of the basal vessels can be found, it naturally varies with the type of patient and with the amount of sclerosis of the basal vessels to which he is subject. In most cases the calcifications are found in persons of rather advanced age.

DR. MARK J. SCHOENBERG: This is an extremely important contribution to clinical ophthalmology. It is a syndrome which has been carefully worked out, and I am sure that we are going to keep it in mind.

I recall eight or nine years ago seeing a case with that syndrome except for the roentgen findings. I referred the patient to Dr. Knapp. He had subnormal intra-ocular tension, cupping of the disk and atrophy. We did not know what to do for him. I wish I could have some roentgenograms taken of him.

DR. ARTHUR M. YUDKIN: I should like to have Dr. Knapp tell more about the vascular changes associated with this syndrome.

DR. SIGMUND AGATSTON: It is interesting to speculate on the etiology of the atrophy. In this form of atrophy there is always a narrowing of the retinal vessels, and it is possible that with this sclerosis there is either a secondary narrowing or actual occlusion of the retinal artery. I saw a case somewhat like this at Bellevue Hospital. The patient had thrombo-angiitis obliterans, and it was believed that sclerosis of the circle of Willis and also an occlusion of the ophthalmic artery were present. One day blocking of the central artery developed. The patient was examined immediately after the accident, and we saw the narrowing of the retinal artery and the interrupted current of blood. Subsequently the retinal artery was very fine in caliber, and optic atrophy developed. Other cases have shown the same thing. In other words, while with sclerosis of the large blood vessels, such as the circle of Willis, the patient does not necessarily have disease of the arterioles of the body, secondary to occlusion of the ophthalmic artery, the retinal artery may become very narrow; that is probably the cause of the secondary sclerosis of the retinal artery, followed by optic atrophy.

DR. KAUFMAN SCHLIVEK: Has Dr. Fineman ever seen these changes roentgenologically without a lesion of the optic nerve?

DR. LEGRAND H. HARDY: To what does Dr. Knapp attribute the cupping of the disk?

DR. ARNOLD KNAPP: These patients have a moderate general arteriosclerosis, without unusual cerebral symptoms. The retinal circulation is practically normal, so that this cannot explain the optic atrophy. Some years ago Dr. Gruening reported a case of wood alcohol poisoning in which the change in the optic nerve was characterized by a deep excavation, as was well shown by a drawing made by Dr. Percy Fridenberg. This suggested that a cupping of the optic nerve head can result from a process which is working from behind, or in the optic nerve itself, without increased intra-ocular pressure. The conditions found in the roentgenogram are not necessarily the direct cause of these symptoms, but indicate a disturbed circulation, with resulting atrophic disturbances, and possibly produce the cavernous atrophy which Schnabel described in glaucoma.

DR. SOLOMON FINEMAN: I believe that one occasionally finds similar calcifications in cases in which optic atrophy is not present, at least not at the time of the examination. Whether these cases represent a precursory stage of optic atrophy, or whether there are other factors which modify the course of the disease, is not known. I do know that occasionally calcified shadows are demonstrable in the orbital fissures and at the base of the sella which resemble calcifications of the vessels of the circle of Willis in persons who do not have clinical signs of optic atrophy at the time of the examination.

DR. ARNOLD KNAPP: Verification rests entirely on the examination of anatomic material, and I should like to urge those who have the opportunity of following cases in old peoples' homes and elsewhere where autopsies are available to be on the lookout for optic atrophy with cupping; the cause of this process can then be determined.

ROENTGENOTHERAPY OF THE PITUITARY GLAND. DR. CLARENCE HARE and DR. CORNELIUS DYKE.

This paper will be published in full in a later issue of the ARCHIVES.

ALGERNON B. REESE, M.D., *Secretary*

December, 1932

MARK J. SCHOENBERG, M.D., *Chairman*

AN OBSCURE DISEASE OF THE CONJUNCTIVA SIMULATING PARINAUD'S CONJUNCTIVITIS. DR. ABRAHAM FINE.

A man, 32 years of age, who admits a syphilitic infection three years ago followed by intensive treatment, has presented since May, 1932, a marked follicular conjunctivitis of the right eye, with enlarged preauricular and submaxillary glands on the right side.

Examination reveals marked edema of the lids and a swelling of the right side of the face. No regional glandular adenopathy is to be seen. On the palpebral conjunctiva, which is congested and hypertrophied, there are large soft granulations, some of them polypoid, which are confined to the tarsal region. The hypertrophy is so marked as to make it difficult to replace the lids, once they are everted. The ocular conjunctiva is edematous and in its lower half covered with granulations. The cornea appears hazy, staining lightly and diffusely with fluorescein and presenting a superficial vascularization in the upper and lower portions.

Smears and cultures at one time revealed xerosis bacilli, and later Staphylococcus aureus. Two guinea-pigs were inoculated with conjunctival scrapings, and are alive and well. Tuberculin tests gave negative results. The Wassermann reaction was four plus in June, but is now negative. Two specimens of serum sent to Washington for agglutination against Bacterium tularense were reported as giving negative results. The biopsy report stated: "ulceration of the mucosa . . . submucosa densely infiltrated with plasma cells and eosinophils . . . no definite giant cells. Diagnosis: chronic granuloma, etiology undetermined." Examination of the blood showed: white blood cells, 12,000; eosinophils, 3 per cent; polymorphonuclears, 69 per cent; lymphocytes, 28 per cent.

Treatment has included the use of: mercurochrome, 2 per cent; applications of copper sulphate and silver nitrate; injections of milk; fourteen injections of arsphenamine and two of bismuth; large doses of potassium iodide; the use of autogenous vaccines, and one radium treatment.

Tuberculosis, I believe, can be ruled out, because of the negative results of the physical examination, tuberculin test, and inoculation of guinea-pigs and because of the failure to isolate the organism in the tissues or on culture. The continued retrogression, despite a Wassermann reaction made negative by treatment, influenced me against the diagnosis of syphilis. Only one case of definite syphilitic granuloma of

the conjunctiva has been reported in the literature (by Goar). After two injections of arsphenamine his patient completely recovered. The staining of excised tissue, according to the technic recommended by Verhoeff, failed to reveal Leptothrix. In the present case, that of a person who has hay fever and in whom a conjunctival infection began in the spring with allergic symptoms, sensitivity to various proteins and eosinophilia, I should like to suggest the diagnosis of allergic conjunctivitis.

DISCUSSION

DR. E. WALDSTEIN: There seems to be a widespread opinion in Germany at present that Parinaud's conjunctivitis and tularemia are identical. For my part, I would think that Dr. Fine's case is either Parinaud's conjunctivitis or tuberculosis of the conjunctiva. The presence of a massive accumulation of Unna's plasma cells would indicate Parinaud's conjunctivitis.

DR. JOSEPH LEVINE: All these tests that Dr. Fine reported were carried out at the Manhattan Eye and Ear Hospital and gave negative results. The report from Washington on the blood was negative for tularemia. We were unable to reach a definite diagnosis in this case.

DR. ABRAHAM FINE: When Parinaud described his three cases he did not present a definite bacteriology or histopathology, and, as a result, opened the door to considerable confusion. Observers describing this disease at a later date thought that tuberculosis was the etiologic factor. Others, when the oculoglandular form of tularemia became a recognized clinical entity, considered this as the probable cause, especially in view of the fact that Parinaud regarded the disease as due to contact with lower animals. Finally, Verhoeff isolated Leptothrix in seventeen of eighteen cases clinically resembling those of Parinaud, and proposed the term "Leptothrix conjunctivae." I think that a better approach to the understanding of this disease would be reached if one used the term "Parinaud's syndrome" and recognized the varied etiology.

MELANOTIC TUMOR OF THE INNER CANTHUS: EXCISION AND ENDOTHERMY. DR. CLYDE E. MCDANNALD.

A man, aged 47, on Oct. 21, 1922, presented at the nasal side of the cornea of the right eye a pigmented tumor which appeared four years previously. In June, 1922, he took a job in a box factory where he was constantly exposed to dust. There was a rapid development of tumor at the limbus, involving the conjunctiva and folding over the cornea. It was not, however, attached to the cornea or sclera, but some pigment was scattered around the growth in the conjunctiva. It was about the size of a large pea.

Complete excision was performed on October 24, using about the technic that might be employed in the removal of a pterygium. Following the operation three applications of radium were made by Dr. John Remer. Thirty milligrams of unfiltered radium, held ten minutes in apposition to the site of the lesion, were applied on three successive dates, November 3, December 4 and Jan. 13, 1923. The tissue was examined by Dr. Dixon, who reported pigmented, round cell sarcoma.

At present there are no signs of recurrence, although the slit-lamp shows some increased vascularization in the region of the scar and scattered pigment dots in the conjunctiva. Vision is 20/15 in each eye.

DISCUSSION

DR. IRA I. KAPLAN: I think that the result is due to the clever operation rather than to the radium therapy, because one would never think of using that amount of unfiltered radium today on ocular tissues, except where one is treating the conjunctiva. That amount of unfiltered radium might have been of service in a case of conjunctival involvement. This case is remarkable for the following reasons: 1. Round cell sarcoma in that portion of the eye is not common. 2. It is a type that can be readily removed. 3. It is remarkable that the eye has remained healthy all this time in spite of the radium therapy, which might have caused necrosis.

ENCAPSULATED HEMANGIOMA OF THE ORBIT REMOVED BY BLUNT DISSECTION. DR. HENRY H. TYSON.

A woman, aged 49, had had a proptosis of 14 mm. for twelve years. From her right orbit there was removed by blunt dissection (without a Krönlein procedure) an encapsulated angioma measuring 37 by 27 by 22 mm. and weighing 304 grains (19.7 Gm.); 4 cc. of blood had been removed from it before operation. Previous to the operation, vision in the right eye was 20/200, with an irregularly enlarged blind spot. Five weeks after the operation, vision was 20/24, and the blind spot almost normal in size and contour. The interesting features of the case were: (*a*) It was demonstrated that moderately large growths may be removed from the orbit without a Krönlein operation. (*b*) As the growth entirely filled the orbit no orbital fat was present; (*c*) Vision was almost restored to normal after the relief of the pressure on, and stretching of, the optic nerve which had been present for about twelve years.

DISCUSSION

DR. JOHN M. WHEELER: I make a very free canthotomy to the periosteum, and then continue the incision at right angles to the canthotomy opening, above and below in the upper and lower fornix. The incision can be made as large as one wishes, and after the healing process has taken place, there is no unsightly scar. How was the preoperative diagnosis of angioma made? Did Dr. Tyson have to cut any muscles in the removal of the growth?

DR. CONRAD BERENS: The patient whom I showed this evening had a smaller encapsulated hemangioma situated near the apex of the orbit between the lateral rectus and the optic nerve. An external canthotomy was performed, and it was necessary to tenotomize the external rectus in order to remove the growth by blunt dissection. The unusual features of the case were: papilledema, anesthesia of the cornea and paralysis of upward rotation of the right eyeball. I could find no reference to anesthesia of the cornea in the cases previously reported. Papilledema was seen by Cresswell and Briggs in a patient who had an encapsulated hemangioma of the orbit. Paralysis of the extra-ocular muscles has

been reported by several observers. Because certain authors still insist on the necessity of performing a Krönlein operation in removing these growths, it is interesting to see the results obtained by more conservative measures.

Dr. Henry H. Tyson: The tentative diagnosis was made by palpation, the growth in the orbit being easily palpable with the fingers. The procedure was the same as Dr. Wheeler followed, but I learned it from the late Dr. Herman Knapp many years ago. I had seen him remove smaller tumors in the same manner. In my case I resected the external rectus muscle. There were no changes in the fundus.

Sarcoma of the Choroid. Dr. John M. Wheeler.

Dr. Wheeler showed a man, 36 years old, who had sarcoma of the choroid in his only seeing eye. In 1926 he had been operated on for glaucoma in each eye. After the operation vision of the right eye was perception of hand movements; that of the left eye, 20/30. The tension remained normal in both eyes over a period of years, and there was no falling off in vision until about August, 1932. At that time he noticed diminution of vision in the left eye, which presented a striking ophthalmoscopic picture. There was a retinal detachment in the lower temporal quadrant, and in the detached area there was a definite solid protrusion with very little pigmentation. Transillumination was interfered with in the area of the protrusion. A diagnosis of leukosarcoma of the choroid was made. The patient received twenty-five roentgen treatments (200,000 volts; 2 mm. copper filter; a distance of 50 cm.). The sarcoma seemed to thrive on the roentgen treatments, and vision diminished from 20/70 on Aug. 10, 1932, to 3/200, with serious contraction of the visual field, on September 29.

The patient objected to the enucleation of an eye that furnished some vision, and it was suggested that the use of radium seeds either outside the sclera or inside the intra-ocular growth might be justified. Reference was made to the experiments of Dr. Foster Moore.

DISCUSSION

Dr. Ira I. Kaplan: I have learned in my work with roentgen rays that ocular structures tolerate them to a marked degree. Seldom does one see any degree of destruction of ocular tissues from roentgen therapy. In the treatment of ocular conditions with roentgen rays the endeavor is to affect not so much the ocular tissue itself as the surrounding area. I believe that the local area is affected only if the tissue is responsive. Embryonic tissue will respond to roentgen therapy, but there are certain tissues which do not respond, and those are made up of the supporting structures, such as are found in some sarcomatous conditions. In lymphosarcoma, in which there is a cellular growth of a more embryonic type, one obtains a response, but with the fibrous and more specialized type there is little response. I hope to establish in the surrounding normal tissues opposition to the further growth of the tumor. I feel that the treatment affects the blood supply surrounding the tumor, making it more difficult for the growth to develop. If radiation is applied to the sclera, it must be filtered sufficiently to pene-

trate in a strength of sufficient degree to destroy the tumor. That is almost impossible without the production of a local ulceration and destruction of the remaining vision. I think that it might be better in this case to open the sclera and implant radium seeds. The advantage of the platinum over the gold seed is the protection to the retina and the surrounding tissues, thus preventing further necrosis with destruction of the eye. I think that would be my procedure in a case like this.

DR. HENRY H. TYSON: I saw three of Mr. Foster Moore's cases. Apparently he inserted the radon seeds through the sclera in some cases; this was followed by a necrosis of from 2 to 3 mm. of the growth around the seed. He also implanted some of them subconjunctivally, and sometimes between the muscle and the sclera. He showed one case of sarcoma which had been apparently cured for two and a half years with no extension or metastasis during that period. I also saw two cases of glioma, in one of which an eye had been enucleated. Radon seeds were inserted through the sclera, and three radon seeds rested on the sclera over the site of the growth. They were allowed to remain about ten days; when too much irritation supervened, they were removed.

DR. CHARLES H. MAY: I saw a case with Dr. Wheeler some time ago which made a great impression on me as to the value of radium. I should like Dr. Wheeler to give the subsequent history of the patient. In the case mentioned—I do not remember whether it was one of intraocular or of orbital tumor—we agreed that it seemed a great pity to sacrifice the eye, and that possibly radium might be useful, provided we obtained consent to the experiment. The parents of the child agreed to what was more or less an experiment, and radium seeds were implanted. I was much surprised when, several months later, examination revealed practical disappearance of the tumor, with recession of the eyeball, so that there was much less proptosis than before. I have not seen the patient for a year or more, and I hope Dr. Wheeler will give the subsequent history, for it was most interesting to see what could be done with radium.

DR. BERNARD SAMUELS: It seems to me that we should take a wider view of this particular case. If the eye is untreated, the patient will be blind; he is almost blind now. If the eye is treated with radium, it will not prevent the onset of blindness. If by a miracle, one were to shrink the tumor by radiation, total and hopeless detachment of the retina would certainly follow. I think that the problem should be presented to the patient for his decision. If he is going to have the tumor taken out in an effort to save his life, now is the time to remove it, and not later on.

DR. ROBERT K. LAMBERT: Two years ago I saw an inadvertent experiment performed, in which radium seeds were implanted in the ethmoid region for nasal polypi. One seed was left in the ethmoid cells within a few millimeters of the optic nerve, as the string was broken off in an attempt to remove the seed. A few months later the patient began to lose vision, and later optic atrophy developed. Whether this was due to the pressure of a foreign body reaction or was a direct effect of the radium is a question. At any rate, the result is suggestive. The case was reported before the Section of Otolaryngology of the Academy by Dr. Kramer.

DR. MARK J. SCHOENBERG: The question comes up whether by manipulating the tumor tissue and introducing radium seeds one may not cause metastases to occur.

DR. JOHN M. WHEELER: Dr. Samuels suggested that the problem be put up to the patient for a decision. I have talked with the patient, and he does not want enucleation. I presume that ought to influence our judgment in handling the case. It seemed to me that in this case we could not promise the patient that his life would be saved by enucleation. He may have changed his mind, but when I last talked with him, he said he would not have his eye removed.

The case that Dr. May spoke of was not one of tumor of the globe, but a neuroma of the orbit, and it did respond miraculously to radium. I saw the patient a few weeks ago, and he showed no signs of recurrence. Te lids and eye were in good condition, and there was no proptosis.

EPITHELIOMA OF THE LID: EXCISION AND PLASTIC REPAIR. DR. WENDELL HUGHES.

This paper will be published in full in a later issue of the ARCHIVES.

MELANOTIC NEVUS OF THE CONJUNCTIVA. DR. GUERNSEY FREY, JR.

A Scotch woman, aged 43, states that there has always been a small pigmented mole on the skin of the left upper lid. Fifteen years ago, during the course of a pregnancy, the pigmented area on the lid enlarged rather rapidly, and then came to a standstill. Seven years ago, a small black spot appeared on the "white of the left eye" temporally; this spot enlarged suddenly during pregnancy, and has been increasing in area during the past four years.

The patient consulted me for the first time on June 25, 1930. There was an area of pigmentation without elevation that involved the skin of the left upper lid adjacent to the free margin, continuing over the margin to the conjunctival surface of the lid, but stopping short of the fornix. There was fairly extensive pigmentation of the bulbar conjunctiva, extending from the limbus temporally, below and nasally, and invading the superficial corneal epithelium for a small area below. These pigmented areas on the lid and on the globe were not continuous. The pigmentation seemed to involve only superficial epithelial cells and did not appear to invade any deeper tissue. Examination of the fundus gave negative results. No palpable glandular enlargement was noted. The patient declined operative intervention.

I have seen her at intervals of about six months since then. On April 2, 1932, there apparently had been no alteration in the lesion. On October 12 the condition was about the same as at present. There has been considerable increase in the area and intensity of pigmentation, with a piling up at the limbus and invasion of the corneal epithelium, causing a reduction of the vision to 20/50. She complained of vague buzzings in the ears and head, which the otolaryngologists believe are of local origin. A complete neurologic examination by Dr. Zabriskie on October 26 did not suggest any intracranial disease. The patient is still opposed to any operation which would involve the sacrifice of the eyeball.

DISCUSSION

DR. CHARLES H. MAY: Not long ago I saw a case in which the growth was not so marked or extensive as this one, but a flat melanoma adjacent to the cornea and about five-eighths by one-eighth inch (1.6 by 0.3 cm.) in size; it was slightly elevated, but did not involve the cornea. This was the type of case in which excision was formerly done with a good deal of damage, but in which we now hesitate on account of the danger of inviting metastases. My attention having been called to the successful use of the thermaphore in a similar case, I made a single application with this heat-generating instrument, using 145 F. for one minute in direct contact after local anesthesia. At the end of from four to five weeks the lesion had completely disappeared.

DR. JAMES W. SMITH: The same evening that Dr. Frey showed his patient here, two years ago, I presented a case of melanoma of the conjunctiva. Five months later the right preauricular gland became enlarged and indurated. The patient was then receiving radium treatment for the melanoma at the Memorial Hospital. A biopsy on the gland showed melanoma. The question of enucleation arose, and Dr. Ewing and a few ophthalmologic consultants agreed that the eye should be removed. The patient had overheard the various opinions expressed, and decided that if he already had an extension to the gland, he might have metastases elsewhere. He refused enucleation, local therapy was continued and the gland was excised. At present he has the full use of his globe, vision of 20/20 and no evidence of metastases.

DR. IRA I. KAPLAN: My experience with melanomas elsewhere has been that no matter what is done, once they are treated, cut out or irradiated, one has trouble; frequently there are metastases to liver and lungs. If something must be done, perhaps it would be well to use roentgen rays; these produce a protective element of vascular closure about the tumor because they act on the endothelium lining the blood vessels and capillaries and form a sort of protective sheath for these vessels, so that the surrounding areas are entrenched against further invasion. Radium has a little different effect than roentgen rays because the gamma ray of radium seems to have some selective action on the pathologic tissues. But melanoma does not consist of the tissue seen in epithelioma, carcinoma or sarcoma, and therefore radium does not affect melanomas as it does other pathologic tissues. I have seen several melanomas that were treated with intensive doses of radium which did not respond at all. I am therefore of the opinion that it is an extremely dangerous practice to use radium in any case of melanoma of the eye.

HEMANGIOMA OF THE CHOROID. DR. F. W. SHINE.

This paper will be published in full in a later issue of the ARCHIVES.

USE OF ROENTGEN RAYS TO CONTROL THE INCREASE OF OCULAR TENSION IN THROMBOSIS OF THE CENTRAL RETINAL VEIN. DR. FRANCIS X. BROPHY.

Following obstruction of a branch of the central vein, it is generally believed that the eye becomes potentially glaucomatous. Thrombosis of

the central vein is said to be complicated by glaucoma in from 20 to 25 per cent of the cases, owing to an exudation of highly albuminous fluid into the ocular fluids. Secondary glaucoma usually does not develop until from six to twelve weeks after the onset of the thrombosis.

The case presented is that of a woman, 47 years of age, under treatment by her physician for increased blood pressure. On Jan. 12, 1932, on awakening, she noticed marked reduction of sight in the right eye. Examination at the Knapp Memorial Eye Hospital ten days later showed the typical picture of thrombosis of the central retinal vein. There were marked swelling and obliteration of the right nerve head, immense extravasation of blood and large exudative areas on the disk and in the retina. Vision was reduced to perception of hand movements throughout the visual fields. The left eye showed considerable angiosclerosis but no hemorrhages or degenerative changes, and vision was normal. The systolic pressure was 220. Twelve weeks after the onset of the thrombosis, the patient returned because of an attack of acute congestive glaucoma in the right eye. The pain was intense, the cornea cloudy, the pupil markedly dilated, the anterior chamber deep and the tension more than 60. Two per cent pilocarpine was used every hour for the next two weeks, but at no time was the tension below 50. Operative intervention was considered dangerous, and Dr. Arnold Knapp suggested the use of roentgen rays. The patient was sent to Dr. Francis Carter Wood about three weeks after the onset of glaucoma, and after the second period of irradiation the patient reported considerable lessening of ocular distress; the eyeball was less congested and the tension was 45. After the third irradiation, she was free from all symptoms, the cornea was clear and the tension was 25. After the fourth irradiation, the tension was 20 and externally the globe had a normal appearance. During this time, the use of pilocarpine had been continued every few hours. The drug had made the pupil almost the size of a pinpoint, without influencing the increased tension. After the fourth period of irradiation, when the tension had been reduced to 20, pilocarpine was instilled twice a day and the pupil widened, but without any return of tension. The use of pilocarpine was then discontinued, and the pupil became moderately dilated.

Ten roentgen treatments were given, at first at intervals of two weeks, then three weeks and finally from four to six weeks. The case has been followed for six months since the return to normal tension; during this time the tension has varied between 18 and 25. The dosage of roentgen rays used was 100 roentgens for five minutes at 140 kilowatts with 4 ma.

The good results obtained with small doses of roentgen rays, if they are applied early, may be due to (1) the direct definite lytic action of the roentgen rays, (2) increased phagocytic action in the thrombus, with disintegration, shrinkage or canalization, or (3) widening of the vessels as part of the hyperemia produced by the roentgen rays, allowing partial circulation and then subsequent changes in the thrombus. It is possible that the thrombus causes only partial obstruction of the lumen, which becomes complete by vascular spasm, and that the roentgen rays, by their hyperemic action, release the spasm, with subsequent changes in the thrombus.

DISCUSSION

DR. HERBERT W. WOOTTON: For some time I have been much interested, mainly from a diagnostic point of view, in these cases of secondary glaucoma of the congestive variety following thrombosis of the central vein. In primary congestive glaucoma one expects a shallow anterior chamber, but of ten or twelve cases following thrombosis which I have observed during the last two years, there was only one in which the anterior chamber was shallow. In several instances it was decidedly deep. I think that this point is of importance in the differential diagnosis. In a case of congestive glaucoma in which the cornea is so edematous that the fundus cannot be seen, and the anterior chamber is deep, the glaucoma is probably not primary but secondary, and one may be fairly sure that it is due to thrombosis of the central vein, assuming that serous cyclitis as an etiologic factor has been excluded.

In regard to the treatment in such cases, I have seen two eyes lost from hemorrhage, following iridectomy. In two other cases, in which the patients declined operation, the pain and congestion subsided after three or four weeks, the tension became normal, and the anterior chamber resumed its normal depth. Both eyes are blind. In a third case with a deep anterior chamber, I attempted to reduce the tension and relieve the pain by repeated paracenteses. The result was similar to that already stated; the pain and tension disappeared and the anterior chamber resumed its normal depth in about four weeks. This eye is also blind. These results make me think that perhaps the use of roentgen rays is no more successful in relieving tension than other methods of treatment or possibly no treatment at all.

DR. MARTIN J. COHEN: I wish to confirm Dr. Wootton's statement with regard to the deep anterior chamber in secondary glaucoma. I recently had a patient, 28 years old, with malignant hypertension and a systolic pressure of 300, who had diabetes and marked vascular changes in one eye. Thrombosis of the central vein developed, the symptoms of which have already been outlined, with marked choked disk, retinitis and all the typical signs of venous compression. A hemorrhage into the vitreous occurred, and at the same time an albuminous exudate appeared in the anterior chamber, causing the lens, iris and suspensory ligaments to be forced back into the vitreous body. The tension in that eye was 75 mm. of mercury (Schiötz). Thrombosis of the central vein developed in the other eye, which showed all the characteristic changes of the fundus of the first eye. A very deep anterior chamber developed, with the same pathologic changes. The tension in that eye was about 60 mm. of mercury (Schiötz). I think that this finding plays an important part in the differential diagnosis between primary and secondary glaucoma.

DR. SIGMUND AGATSTON: There are many cases of thrombosis of the retinal vein, some partial and others total. I should like to know how irradiation affects the thrombosis. It seems to me that in complete thrombosis of the central vein the chances of return of vision are practically nil. I do not see how vision can return if there is a complete thrombosis of the main branch of the central vein, as there is no possibility of collateral circulation.

DR. IRA I. KAPLAN: It is interesting to see the favorable results in some cases following irradiation. I sometimes think that the curative effects are produced by nature itself rather than by radiation. In certain conditions, however, hyperemia relieves tension merely by increasing the tension at certain times. I doubt whether there is a definite obstruction to the central vein in the case described. I believe that it is correct to state that if a permanent obstruction to the central vein and a sufficient destruction of the retina occur, there will be no return of sight. But this thrombosis may not be permanent. The hyperemia may have increased the width of the blood vessels so as to allow a moderate passage of blood around the thrombus, to keep up the feeding of the retina until the collateral vessels take care of the blockage. Roentgen therapy increases the hyperemia, causing a response in the blood cells, with an increased leukocytosis. This leukocytosis is of the same type as that occurring in response to injury; it is the ambulance corps of the blood system which fights infection.

DR. FRANCIS X. BROPHY: Loewenstein and Schnyder have stated that the veins are at first occluded, since digital pressure is unable to empty the veins in the early stages. After irradiation this digital pressure will cause a collapse of the veins, showing that the circulation has been restored.

EXOPHTHALMOS FROM ORBITAL EXTENSION OF EPIDURAL ABSCESS. DR. EDWARD B. GRESSER.

A Negress, aged 42, complained of a periodic prominence of the right eye for the preceding six months. It occurred for periods of about two days and was not accompanied by symptoms. For six weeks prior to admission the protrusion had remained and progressed, and for the past two or three weeks the eye had been red and painful, and vision had been impaired. The right globe was so proptosed that the lids could not be closed over it. The cornea was opaque and ulcerated. The left eye showed no abnormality.

All laboratory tests gave negative results except that the blood sugar amounted to 307 mg., and the roentgenogram of the skull showed unusual thickening of the inner table of the vault in the frontal region and extensive destruction of the posterior, superior and inner wall of the right orbit and right ethmoid cells. The preoperative diagnosis was orbital tumor.

An exenteration was attempted during which pus was encountered. An enucleation was performed, and the periosteum of the orbital roof was incised, which freed some greenish, odorless fluid under tension. The area containing fluid pulsated and was found to be situated between the dura and orbital periosteum. Culture of the pus showed Staphylococcus aureus.

IRRADIATION IN OCULAR CONDITIONS. DR. IRA I. KAPLAN.

Irradiation is used (1) as a preoperative measure for conditions in and about the eye, (2) as a postoperative measure for malignant conditions following removal of the eye and (3) in the treatment of surface lesions in and around the eye.

Most patients, when they reach the therapist, either have been operated on or have advanced malignant lesions. One is seldom asked to treat conditions with irradiation which are not associated with advanced malignancy. With irradiation, we hope first to destroy the pathologic tissue; second, to create in the normal surrounding tissue a defense against, and restriction, of further malignant growth, and third, to stimulate repair. We make equal use of both roentgen rays and radium, choosing the therapy which we think appropriate, and we obtain good results with both when they are properly used. To be effective, radiation must be properly applied in the first instance, since repeated treatments may lead to severe necrosis and destruction. Excessive treatment of lupus of the nose may lead to intractable ulceration and the establishment of a malignant lesion invading the orbit. Irradiation is the method of choice in the treatment of surface epitheliomas.

Book Reviews

Behandlung der Netzhautabhebung mit multipler diathermischer Stichelung. By Dr. K. Safar, Vienna. Price, 9 marks. Pp. 162, with 12 text illustrations and 3 colored plates. Berlin, S. Karger, 1933.

Professor Meller, in the preface of Dr. Safar's monograph, speaks of the importance of Gonin's teaching that closure of the retinal tear is essential for the recovery of the detachment, and then traces the development of the diathermic treatment by the contributions of Larsson, Weve and Safar. Meller is convinced of the great value of this procedure and of its safety to the eye.

The author begins with a consideration of the causes of detachment of the retina. He believes that in most cases the cause is a tear in the retina which permits the entrance of vitreous fluid underneath the retina, resulting in retinal detachment. Sometimes as late results of injuries detachments develop from the contraction of cicatricial scars in the retina, along its inner surface or in the vitreous, which then lead to a rupture and the exit of the vitreous fluid. These contracting bands can also develop from the organization of hemorrhages or exudates after injuries, or from endogenous causes. The explanation for the frequency of detachment in myopic eyes is to be found in degenerative and atrophic changes which occur in the retinas of these eyes, the so-called cystoid degeneration, which occurs in the periphery of the retina and leads to changes that facilitate tears. Vogt has described similar changes in senile eyes, and Meller has drawn attention to tears in the retina which occur in the vicinity of a choroidal focus where the retina is thin.

The percentage of cases in which tears have been found is greatest when the condition is recent, which rather suggests that the rupture precedes the detachment. Sometimes the holes are small, and in the presence of cloudy media, of a pupil that cannot be dilated or of nystagmus, the tears are discovered only with great difficulty. In these cases the location of the detachment and the history of the onset of the disturbance of the visual field suggest the site or the beginning of the detachment and the probable location of the tear. The large tears following trauma are difficult to cure with conservative measures, and often operative treatment with ignipuncture is not satisfactory on account of the localized action of the cautery. These cases are not, however, unfavorable for multiple electrocoagulation, particularly if the tear is situated below, where the retinal detachment remains limited for months. The search for tears requires great patience and practice, particularly when the tears or perforations are situated in the periphery. Under these conditions the dilatation has to be maximal, as is best obtained with the use of epinephrine, and when the media are cloudy an intense light like Lauber's lamp will be of advantage. While in Gonin's operation the exact determination of the position of the tear is essential, this is not so necessary with multiple diathermic punctures, though it is desirable to

have a clear idea of the location in order to operate in as small an area as possible, and not to injure the other parts of the fundus.

The author has found that Lindner's method with the perimeter-Gullstrand to localize the tear according to meridian and latitude is sufficient, and then the distance from the limbus is measured on the scleral surface by means of calipers. If the tear is situated very far back, an exact localization is possible only if the retinal detachment is shallow. If there is a large cystic degeneration it is often well to allow the fluid to escape from the subretinal space by a preliminary diathermic puncture. Then when the retina has flattened out, it is much easier to determine exactly the location of the tear. Here, again, the ophthalmoscopic control during the operation is important.

The author reviews the previously used methods for the treatment of retinal detachment, and then describes the operation with diathermy.

After the publications of Weve and Larsson, Meller showed that unexpected reactions in the form of opacities of the vitreous and an injury to the inner membranes of the eye far beyond the area that was treated do occur, so that notwithstanding an adhesion of the retina vision was so damaged that no result was obtained. These results were undoubtedly due to an overdose of the current and the use of too large electrodes. It occurred to the author that these dangers could be avoided and a broad adhesion obtained without damage to the tissues, particularly to the retina, if multiple diathermic punctures were applied cautiously with needles which puncture only as far as the subretinal space. Weve uses a thick or a fine needle which he introduces up to the retina. This needle is repeatedly introduced while the area is being kept under observation with the ophthalmoscope.

Safar's method leaves the retina and the vitreous alone as much as possible; the short needle is introduced as far as the subretinal space. The use of the high frequency current will allow penetration of the sclera and choroid with simultaneous coagulation of the choroid. Then, after the escape of the subretinal fluid, the intact retina comes to adhere to the coagulated choroid. The retina and the tear are untouched. The vitreous is shut off by adhesion of the retina to the choroid surrounding the tear.

Technic.—The author then describes the preoperative preparation of the patient and the steps of the operation with exposure of the sclera and the insertion of the electrodes.

Safar's electrodes are 1.5 mm. long, so that too deep a penetration into the interior of the eye is prevented. They come singly, or grouped in three, five and seven prongs, either on a straight or on a curved handle; the one or the three pronged instruments come also without handles, so that they can be left in situ during the operation.

Safar's principal contribution to the endothermic treatment of detachment has been the use of the fine point electrodes which, as they can be retained in place, prevent the subretinal fluid from escaping, and the retina is protected from injury during the application of the other electrodes. Selection of the electrodes depends on the site of the tear. For tears in the periphery, or for any tears that are accessible from the front, he likes to use the pin electrodes to cause coagulation at the proximal margin of the affected area. In addition, the area lying more anteriorly is treated with the multiple or single electrodes. In the

latter position, the escape of the subretinal fluid does not cause any particular damage. In cases of retinal detachment without visible tear, it is necessary to treat from one fourth to one half of the periphery of the eyeground, which includes that part of the retina where the detachment has apparently begun and where the tear should be situated. Tears situated far posteriorly are treated with the seven-toothed electrode on a curved handle at one operation, and no damage to the macula has been observed. It is essential that the subretinal fluid escapes, and that the detached retina is capable of uncurling; hence, recent cases do well, even if the detachment is pronounced. It is not well to wait if the macula is also involved.

Aphakic eyes in general give a bad prognosis, though sometimes even patients with such cases may be operated on with success if the detachment is flat and not too old. Postoperative vitreous opacities and hemorrhages were observed in only one case, while small hemorrhages lying at the margin of the choroidal areas are not uncommon.

The result of the coagulation is an adhesive choroiditis which runs its course after six or eight weeks. The ophthalmoscopic picture is that of an old choroiditis with areas of atrophy and pigment displacement; nodular, thick scars and connective tissue formation in the retina and vitreous as they are observed after Gonin's operation through the action of the thermocautery or as the result of severe hemorrhages do not occur, particularly if one does not puncture into the vitreous.

Results.—Thirty-three patients were operated on without previous selection, and every patient for whom there was the slightest possibility of improvement was accepted. The fresh cases nearly always gave satisfactory results. Patients with cases of a duration of months and years were also operated on, and if the detachment was not total and the retina was not contracted, remarkable improvement in function was observed. More than half of the patients who were operated on by diathermy were unsuited for the Gonin operation according to indications set by Amsler, and a certain number of them had favorable results.

A full description of these cases is given, with the following results: Of thirty-four eyes, the retina became replaced in twenty-one. The ages of the patients varied between 12 and 81. The successful cases were practically all recent ones. Tears and holes in detached retinas were found in sixteen of the twenty-one successful cases.

The author further divides the cases into those of: (*a*) myopia, eighteen cases, in which fifteen were healed and three unaffected; (*b*) traumatic detachment in nonmyopic eyes, five cases, with four healed and one unaffected; (*c*) detachment in aphakic eyes, seven cases; one patient was cured after two operations, two were much improved, and in four there was no result; (*d*) retinal detachment from other causes, four cases; one patient was cured, and three had no result.

Postoperative complications consisted in glaucoma in one case and vitreous hemorrhage in one.

Histologic examination could be made of the eyes of two patients who were operated on with electrocoagulation; the patients died of heart disease. The microscopic changes are described and were found similar to those obtained experimentally in animals' eyes.

A summary giving the principal points in the method and the results concludes the monograph. Six excellent drawings in colors show a case

of ora tear before and after operation, examples of degeneration of the retina and choroidal areas as they appear surrounding a tear after successful operation.

Dr. Safar has written a thorough description of electrocoagulation. This method constitutes a great advance in the operation for retinal detachment, particularly when Safar's ingenious electrodes are employed; its technic is simple, and is the least dangerous to the eyes of the operations so far devised for retinal detachment. A. K.

Injuries of the Eye: Diagnosis and Treatment, Forensic Procedures and Visual Economics. By Harry Vanderbilt Wuerdemann, M.D., ScD., F.A.C.S., Colonel, Medical Reserve Corps. Second edition. Price, $13.50. With 236 illustrations and 10 colored plates. St. Louis: C. V. Mosby Company, 1932.

The field indicated by the title of this volume is one of increasing extent and importance in which the author has been an able worker for many years. It is connected with, or has connotations and implications in, many domains not only of medical pathology, diagnosis and treatment, but of social and industrial economics and technology as well. An inclusive and detailed consideration of all these aspects might well require the collaboration of a number of workers and produce finally an encyclopedic system or reference handbook on the subject. This is, for the present, a pious wish. Wuerdemann has not attempted the impossible. Aiming to give "an exhaustive and authoritative work upon Injuries of the Eye," he has largely rewritten and added to the matter contained in the first edition (1911) of his work. In this practical purpose he has succeeded. Part one, dealing with general injuries, considers the types of ocular traumatism, their etiology, mechanism and complications, as well as the principles of diagnosis, prophylaxis, conservation and operative treatment. Part two considers the injuries of the various ocular structures, in their order, as well as those of the visual nervous system and of the lacrimal apparatus, lids and orbit. This arrangement is eminently practicable and of special value in looking up data about a given form of traumatism in an individual case. It has, however, the disadvantage of repetition, bringing with it a tendency to neglect certain details or at least not to go into them sufficiently. This criticism applies to the sections on the mechanism of injuries and the protection afforded by nature, which might well have been amplified and, above all, illustrated. Part three deals with forensic medicine and takes up the physical examination of the patient, malingering, self-inflicted damage, expert ophthalmologic evidence, protective legislation and court decisions, as well as the methods for the scientific estimation of economic damage according to Magnus-Wuerdemann. Despite his manifest leaning toward this method, the author thinks "that the U. S. Government schedules, which agree, in the main, with those of foreign governments should be accepted generally as a simplified system." This code might to advantage have been incorporated in a reference work of this kind which should also have given one or the other of the more or less official tables for the estimation of loss of economic vision (sight, motility, field). Where so much that is good is presented and all is presented so clearly, it seems invidious or carping

to refer to this or that "relative insufficiency" or to the omission of a name, a symptom, a procedure, here or there. The more so as these defects are generally situated in the far periphery of practical ophthalmology. They do not interfere with a clear view of the important picture or with insight into essential matters. The illustrations are of unequal value. The photographs, especially those of plastic operations and of pathologic sections, are excellent. The pictures of the fundus are good; the original drawings are instructive and accurate, but not distinguished technically. The presswork is commendable except for the use of a calendered paper and some flaws in the index. For the record and possible future inclusion, we note the omission of Wheeler's operation for restoration of the socket, glaucoma after acid burns, Purtscher's Fernschaedigung (of the retina), retrobulbar or subcapsular (Tenon) insufflation of air as an aid in roentgen localization of foreign bodies and serotherapy (especially preventive inoculations and nonspecific protein therapy). Fuller details of plastic procedures on the lids and conjunctiva, on ligation in pulsating exophthalmos and on the use of the slit-lamp would have been valuable.

PERCY FRIDENBERG.

Orthoptic Training with the Stereo-Orthopter. Cloth. Price, $1. Pp. 76. Southbridge, Mass.: American Optical Company, 1933.

This publication is apparently written by the scientific staff of the American Optical Company. The opening chapters give a brief résumé of the history of physiologic optics, the types and methods of diagnosis of various types of amblyopia, the principles of amblyopic training, the development of binocular vision and the types of squint which can be relieved by orthoptic training.

The authors then describe the stereo-orthopter, which differs from other orthoptic instruments in using mirrors instead of prisms (to eliminate aberration), in having a wide range of adaptability to all degrees of squint, in combining amblyopia, suspenopsia, innervation and fusion exercises into one and in being automatic in operation.

A short description is given of the method of using the apparatus. It is impossible to judge the efficiency and ease of operation of the apparatus from the description alone.

In the six cases described, the patients seem to have responded remarkably well to the treatment. There is no mention of the percentage of patients treated who were improved.

I believe that a cooperative patient is of more importance in orthoptic training than impressive appearing apparatus. Hence, I believe that comparison of this instrument with the simpler types of apparatus at present in use is necessary before any definitive statements can be made as to the value of the stereo-orthopter.

W. F. DUGGAN.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Prof. F. de Lapersonne, 217, Faubourg St. Honoré, Paris.
Secretary-General pro tem.: Dr. A. Churchill, 66, Boulevard St. Michel, Paris.

International Ophthalmologic Congress

Secretary: Dr. Francisco Poyales, Olozaga 3, Madrid.
Place: Madrid. Time: April 16-22, 1933.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Sir J. Herbert Parsons, 54 Queen Anne St., Cavendish Square, London, W. I.

Ophthalmological Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgoam Road, Bombay 4.
Secretary: Dr. C. R. Athawale, Bhatwadi Girgoam, Bombay 4.
Place: A. M. C. Free Ophthalmic Hospital Bldg., Poi Bawadi Parel, Bombay 12.
Time: First Friday of every month.

Ophthalmological Society of the United Kingdom

President: Mr. A. H. H. Sinclair, 6 Charlotte Square, Edinburgh.
Secretary: Mr. Humphrey Neame, 56 Portland Place, London, W. I.
Place: London. Time: May 11-13, 1933.

Oxford Ophthalmological Congress

Master: Dr. Cyril Walker, Bristol, England.
Secretary: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. A. C. Hudson, 50, Queen Anne St., W. 1, London.
Secretary: Dr. J. D. Magor Cardell, 27, Weymouth St., W. 1, London.
Place: London. Time: June 9, 1933.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.

NATIONAL

American Medical Association, Section on Ophthalmology

Chairman: Dr. Frederick H. Verhoeff, 82 Commonwealth Ave., Boston.
Secretary: Dr. Parker Heath, 1551 Woodward Ave., Detroit.
Place: Milwaukee. Time: June 12-16, 1933.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
President-Elect: Dr. J. M. Wheeler, 30 W. 59th St., New York.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Boston. Time: Sept. 18-22, 1933.

American Ophthalmological Society

President: Dr. Thomas B. Holloway, 1819 Chestnut St., Philadelphia.
Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.
Place: Washington, D. C. Time: May 8-10, 1933.

SECTIONAL

New England Ophthalmological Society

President: Dr. Hugo B. C. Riemer, 128 Newbury St., Boston.
Secretary-Treasurer: Dr. Benjamin Sachs, 483 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Hans Barkan, 490 Post St., San Francisco.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: San Francisco. Time: June 28-30, 1933.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. R. Secoy, 1802 Hewitt Ave., Everett, Wash.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. R. B. Smith, Alma, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. John B. Gregg, 101 S. Main St., Sioux Falls, S. Dakota.
Secretary-Treasurer: Dr. F. H. Roost, 420 Trimble Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Secretary: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
Place: Richmond, Va. Time: November, 1933.

Southwestern Michigan Triological Society

President: Dr. W. E. McGarvey, 802 Jackson City Bank Bldg., Jackson.
Secretary: Dr. Don C. Lyons, 1405 Union and Peoples National Bank Bldg., Jackson.
Time: Third Thursday of alternate months.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Ralph W. Danielson, 258 Metropolitan Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. J. J. Cohane, 59 College St., New Haven.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. Thomas H. Hall, Jr., 617 Mulberry St., Macon.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. Robert J. Masters, 805 Hume-Mansur Bldg., Indianapolis.
Secretary: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: December 13, 1933.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. W. W. Pearson, 410 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. W. F. Boiler, 105 E. Iowa Ave., Iowa City.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Ray Connor, 28 W. Adams Ave., Detroit.
Secretary: Dr. Ralph B. Fast, 136 E. Michigan Ave., Kalamazoo.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Harold J. Rothschild, 239 Lowry Bldg., St. Paul.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Lawrence G. Dunlap, 101 Main St., Anaconda.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.
Place: Anaconda. Time: July 11, 1933 (meetings are held semi-annually).

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Charles H. Schlichter, 556 N. Broad St., Elizabeth.
Secretary: Dr. B. E. Failing, 31 Lincoln Park, Newark.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. D. F. Gillette, 109 S. Warren St., Syracuse.
Secretary: Dr. Frank M. Sulzman, 1831 Fifth Ave., Troy.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Axel Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

President: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Secretary: Dr. N. A. Bolotow, 108 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Walter Bristow, Medical Bldg., Columbia.
Secretary: Dr. J. W. Jervey, Jr., 101 Church St., Greenville.
Place: Columbia. Time: October, 1933.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. G. Reaves, 422 W. Cumberland Ave., Knoxville.
Secretary-Treasurer: Dr. Ralph O. Rychener, 1720 Exchange Bldg., Memphis.
Place: Nashville. Time: April 10, 1933.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: O. S. Hodges, 388 Pearl St., Beaumont.
Secretary: Dr. A. F. Clark, 1034 Nix Professional Bldg., San Antonio.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. T. Hasler, 290 W. Center St., Provo.
Secretary-Treasurer: Dr. T. F. Welsh, 920 Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Frank P. Swart, 142 W. York St., Norfolk.
Secretary-Treasurer: Dr. H. G. Preston, 307 Professional Bldg., Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.
Secretary: Dr. Harry V. Thomas, 95 Fairmont Ave., Fairmont.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Elbert A. Curtis, 65 Central St., Newark.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ACADEMY OF MEDICINE OF TORONTO, SECTION OF OPHTHALMOLOGY

Chairman: Dr. P. J. F. Houston, 34 Hurndale Ave., Toronto.
Secretary: Dr. Alexander E. MacDonald, 151 Bloor St., W., Toronto.
Time: First Monday of winter months.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. G. E. Clay, 384 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Harry Friedenwald, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. William M. Rowland, 906 Cathedral St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. W. F. C. Steinbugler, 515 Ocean Ave., Brooklyn.
Secretary: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. H. D. Andrews, 33 Allen St., Buffalo.
Secretary-Treasurer: Dr. Thurber Le-Win, 112 Linwood Ave., Buffalo.

Chicago Ophthalmological Society

President: Dr. Dwight C. Orcutt, 30 N. Michigan Ave., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medical and Dental Arts Club. Time: Third Monday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. M. W. Jacoby, 2323 Prospect Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 1065 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. Roy B. Metz, Hanna Bldg., Cleveland.
Secretary: Dr. Paul G. Moore, Medical Arts Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. L. F. Appleman, 308 S. Sixteenth St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. John B. Alcorn, 167 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Edward W. Harris, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. O. M. Marchman, 1717 Pacific Ave., Dallas, Texas.
Recording Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Corresponding Secretary: Dr. J. M. Potts, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. Cecil C. Jones, 1121 Equitable Bldg., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Raymond S. Goux, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. R. H. Seeley, 9 Court St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Valin R. Woodward, Flatiron Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
Place: Medical Hall, Medical Arts Bldg. Time: 7.30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Carl F. Snapp, 26 Sheldon Ave., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 320 Medical Arts Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. J. F. Gamble, 201 Main St., Houston, Texas.
Secretary: Dr. B. F. Payne, 1304 Walker Ave., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Albert N. Lemoine, 906 Grand Ave. ,Kansas City, Mo.
Secretary: Dr. Harold B. Hedrick, 618 Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Ferris L. Arnold, 923 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Clyde E. Harner, 312 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from September to June.

Los Angeles Society of Ophthalmology and Oto-Laryngology

Acting President: Dr. D. E. Godwin, 910 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. F. H. Brandt, 1052 W. Sixth St., Los Angeles.
Place: Elks Temple. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. Karl N. Victor, Breslin Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Place: French Village. Time: Second Thursday of each month, September to May, inclusive, at 6:30 p. m.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. Albert P. Tibbets, 1801 I St., Washington.
Secretary: Dr. G. Victor Simpson, 1710 Rhode Island Ave., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month. Annual meeting, Jan. 10, 1933.

Milwaukee Oto-Ophthalmic Society

President: Dr. Samuel G. Higgins, 324 E. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Montreal Ophthalmological Society

President: Dr. J. P. E. Bousquet, 66 Sherbrooke St., West, Montreal, Canada.
Secretary: Dr. J. A. MacMillan, 1410 Stanley St., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Eugene Orr, 706 Church St., Nashville, Tenn.
Secretary-Treasurer: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. Charles A. Bahn, 150 Baronne St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Mark J. Schoenberg, 1160 Park Ave., New York.
Secretary: Dr. Algernon B. Reese, 73 E. Seventy-First St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.
Annual Session: April 17, 1933.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. L. G. Howard, 532 First Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. W. A. Cassidy, 1620 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. R. W. Vaughan, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. W. E. Munroe, 277 Alexander St., Rochester, N. Y.
Secretary-Treasurer: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Max W. Jacobs, Missouri Theatre Bldg., St. Louis.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Charles J. Boehs, Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. George N. Hosford, 490 Post St., San Francisco.
Secretary: Dr. Russell Fletcher, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. T. D. Boaz, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. David Hartin, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. W. W. Henderson, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. W. G. Dickinson, 504 State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. Harold H. Joy, 504 State Tower Bldg., Syracuse, N. Y. N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy Dunlap, Medical Arts Bldg., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, Medical Arts Bldg., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.